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Vol. 10, No. 1, May, 1955

The Incidence and Management of Fractures of the Neural Arch

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During surgery for the so-called disk syndrome over the past 10 years I have repeatedly found an unusual condition involving the laminae. In elevating or stripping the tissues away from the lamina, I have noticed that my elevator would strike a roughened area, after which I would lose the sensation of the blade gliding over a smooth surface. On examination I have found that the cortex was missing from a portion of the posterior surface of the lamina. The dropping of all or a portion of the blade of the elevator into this area was the reason for the change in action of the elevator.

This defect had no set pattern. It might be a long narrow area running transversely or obliquely across the lamina, or it might be of a wide irregular shape. The lamina under this area was of a soft friable consistency which was easily cut with a rongeur or punch. In several cases I was able to remove it with a periosteal elevator.

In each case, the remainder of the involved lamina had a normal cortex and was of normal consistency. It offered the normal amount of resistance to the rongeur or punch. In each case there was a marked hypertrophy of the ligamentum flavum.

Immediately after surgery, the x-rays of the lumbar area were reviewed with Dr. T. C. Hobbs, Chairman of the Radiological Department at Doctors Hospital. In no case were we able to pick up any defect in the x-rays. On several occasions Dr. Hobbs was called to surgery to view the area.

In 1948[1] I wrote of this condition suggesting that I was of the opinion that this was the aftermath of a fracture of a lamina. This was based on fifteen cases Dr. Donald Siehl and Dr. W. G. Bradford had seen while operating with me. Needless to say, this aroused much controversy.

Review of the literature available on this subject was of no help. Fracture of the lamina was not even mentioned in most cases. If it was mentioned, a single line stated that it was sometimes found in conjunction with extensive vertebral fractures. Neurosurgeons who were interviewed were unable to explain the condition. However, periodically another one of these cases would come to attention in surgery, and the search would start all over.

In the preparation of this paper fifty records of cases of disk syndrome were reviewed. Of the fifty cases picked at random, fourteen had this condition I have just described. Five involved the right and nine the left lamina. Laminae of the third, fourth, and fifth lumbar vertebrae were involved. In no case were two found to be involved.

Preoperative x-rays showed incomplete fusion of the neural arch in six cases. All involved the lamina. Seven showed narrowing of the lumbosacral interspace, and there was one fractured articular facet. Each of the cases involving the fifth lumbar vertebra had a traumatic background.

During the past year, as new editions of the various books on orthopedic, traumatic, and neurologic subjects have been released, I have found that considerable attention is being given to fractures of the various components of the neural arch. I have studied these texts and have culled from them the latest thinking on this particular condition.

In considering the various causative factors of the so-called disk syndrome one must not lose track of the fact that injuries to the various components of the neural arch play a very important part. It is necessary also to consider the functions of the constituent parts in injuries to the vertebrae. A vertebra is made up of: (1) A columnar body, situated anteriorly and ventrally, whose function is to support weight. (2) A vertebral arch made up of two pedicles and two laminae placed behind the body. With the body, the neural arch encloses the vertebral foramen. Collectively the vertebral foramina form the vertebral canal enclosing the spinal cord. The function of the vertebral arch is to provide protection to the cord. (3) Three processes, two transverse and one spinous, which afford attachments to the muscles. Their function is to provide movement. (4) Four articular processes, two superior and two inferior, which project up and down respectively from the arch and come into apposition with the corresponding articular processes of the vertebrae above and below. Their function is to restrict motion in certain directions and to prevent the vertebrae from slipping forward.[2]

The intervertebral foramen results from the juxtaposition of a superior and an inferior vertebral notch, bounded above and below by pedicles, anteriorly by the intervertebral disks and parts of the two vertebral bodies it unites, posteriorly by the superior and inferior articular processes, and laterally by the ligamentum flavum. The function of the intervertebral foramen is the transmission of the spinal nerve and its accompanying intervertebral vessels. Narrowing of this foramen makes it easy for the nerve root to be traumatized. The resultant effect is indistinguishable from that of protrusion of the disk.

At the time of injury, it is difficult to manipulate the patient sufficiently to accomplish the varied projections necessary for a complete x-ray examination. Oblique studies are indicated, and every effort should be made to rotate the patient carefully into the correct position to obtain them along with the routine anteroposterior and lateral studies. Without oblique projections it is almost impossible to analyze the laminae and articular facets, which are so often involved.

When confronted with the possibility of a fracture of the spine, the roentgenologist and orthopedist should follow a definite routine of analysis of the x-ray film.[3] (1) The over-all alignment should be considered. (2) Each component of all vertebrae should be outlined and the alignment of one portion considered with respect to the succeeding portion; careful consideration should be given to the contours, and any evidence of irregularity searched for. (3) Evidence of spacing variations between successive bodies and successive articular processes should be looked for. (4) The relative dimensions of each portion should be compared with those of the preceding and succeeding vertebrae. (5) The osseous structures should be examined for any defects.

When violent trauma has been the causative factor, consideration should be given to the physiologic

aspects of anatomy. The physician should correlate any one defect with other possible damage which might not be evident in the initial studies.

A good example of this is that the fulcrum of articulation of the vertebrae is at the plane of the nuclei pulposi. Hyperflexion causes most fractures of the vertebrae. Therefore, not only an anterior or lateral wedging may result, but there may also result a subluxation or dislocation of the articular facets, and, possibly, fracture of either or both laminae and laceration of the interspinous ligament.

1. If a slight anterior wedging of the body is found, slight subluxation of the articular facets should be looked for.

2. In fracture-dislocation, the physician should search for fractures of the laminae or complete dislocation of facets.

3. In a single laminar fracture, fracture of a transverse process may also occur.

4. In a transverse process fracture, fracture of laminae should be looked for, especially when the fourth and fifth lumbar vertebrae are involved.

De Lorimier, Moehring, and Hannan[3] report that 80 to 90 per cent of all vertebral fractures occur in the body; otherwise the laminae, the articular processes in the region of the interarticular isthmus, the transverse processes, or the spinous processes are involved. Fracture of the pedicle rarely occurs.

The examiner must not lose sight of the fact that congenital defects and failure of fusion of the pedicle, isthmus, or the lamina are sometimes mistaken for traumatic defects. They are capable of producing definite symptoms. These lesions are most frequently seen in the lumbar or lumbosacral area of the spine.

It is not infrequent to find compression fracture of a vertebral body associated with fracture of an articular process, lamina, or spinous process.[4] Usually little or no separation of the fragments results from these associated fractures, but extension hemorrhage into the area may occur. Occasionally pressure on the cord or nerve roots is caused by one of the fragments or the displacement resulting from the fracture. When this occurs it is usual to find the interspinous ligaments and the ligamentum flavum badly torn.

Howorth[4] reports that while doing spinal fusions for compression fractures of the vertebral body he has found associated fracture of the posterior elements in 15 per cent of his cases. Most of these were not visible in the films even when the films were reviewed at operation. At surgery he sometimes found several fractures of the posterior elements when only one had been seen on the film. In severe compression fractures associated with fracture of the laminae or the articular processes, spinal fusion is the treatment of choice. At the same time, pressure against the dura, spinal cord, or nerve root may be dealt with and relief obtained.

Isolated fractures of the articular facets are difficult to diagnose.[5] Fractures of this type may cause severe local and referred pain along with limitation of motion. Suspicion of such a fracture is aroused by localized pain following a spinal injury after other gross lesions of the spine have been eliminated.

Treatment consists of plaster fixation for a period of 10 to 12 weeks. This is usually sufficient in early, uncomplicated cases. When the diagnosis has been made late or the symptoms do not subside under rest and supportive treatment, fusion of the articular facets is advisable.[6]

Mensor's[7] studies indicate that fractures of the articular processes are of relatively frequent occurrence and that greater interest in a more careful routine investigation of these portions of the vertebral column should be insisted upon.

Fractures of the articular processes manifest themselves as irregular linear areas of rarefaction extending transversely or obliquely across the processes. They are usually associated with fractures elsewhere in the vertebra. They must be distinguished from congenital, persistent separation of the epiphysis, which is frequently seen at the tips of the inferior articular processes in the lumbar region. Early diagnosis and the institution of adequate conservative treatment give an excellent prognosis for complete recovery.

Fractures of the laminae may be caused by direct blows or falls but are more frequently produced by hyperextension and sudden severe muscle contractions. Congenital nonfusion of a lamina is relatively common, especially in the lower lumbar and upper sacral regions.

Garland[8] emphasizes the bony variations and anomalies at the lumbosacral region consisting chiefly of variations in the shape and size of the transverse, articular, and spinous processes. The differential diagnosis between anomaly and fracture is difficult. Acquired traumatic lesions show definite marginal changes with the passage of time. Meticulous comparison of good roentgenograms made at intervals of 4 or more weeks will usually serve to differentiate congenital from acquired lesions.

Direct violence from the rear against the spinous processes may fracture one or both laminae and compress the posterior arch sufficiently to cause direct cord pressure and paralysis.

The ligamenta flava are composed of yellow elastic tissue and extend between the laminae across the spinal canal.[9] The two ligaments of each interlaminar space help make up the lateral margins of each intervertebral foramen. When rupture or other injury occurs to the ligamentum flavum, it may hypertrophy and thereby compress the corresponding roots of the spinal cord.

Hypertrophy of the ligamentum flavum was once a very popular diagnosis as the sole cause for nerve root compression. It is now felt that this condition is only a part of the over-all picture. Removal of the ligamentum flavum to obtain operative exposure of the nerve root and the disk plays an important part in decompression of the nerve root.

Thickening of the ligamentum flavum has been reported in a large per cent of proved cases of herniated intervertebral disk.[10] This condition rarely occurs without protrusion of the disk. The finding of such a hypertrophy should lead to a careful search for a protruded underlying disk.

In closing I would like to emphasize that fractures of the lumbar vertebrae, either those of the bodies or other elements, may cause low-back pain and sciatic radiation. One should remember, however, that injuries of sufficient severity to fracture any portion of a vertebra may also injure the intervertebral disk.

117 W. Third Ave.

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Sclerosing Nonsuppurative Osteomyelitis
(Sclerosing Osteitis of Garré)
Case History

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INTRODUCTION

The patient, a 37-year-old white plumber, was first seen on November 23, 1953, in consultation at the office. His chief complaint was of pain in the left heel. The onset of the pain was about 1 year prior to the consultation. There was no history of trauma or injury. The patient stated that the area was "sore as a boil," and he went on to describe pain typical of a boil prior to incision and drainage. He had consulted various doctors who had prescribed numerous arch supports and conservative therapy, but there had been only slight improvement. The pain occurred mainly on pressure, such as bearing weight. However, the patient always had some pain that was throbbing in character. The pain was slightly more pronounced when he began to walk than after he had taken a few steps. For the previous several weeks he had noticed a bump growing on the superior lateral aspect of the left calcaneus. He had noticed no other swellings on his body.

HISTORY

The patient's mother had had arthritis and had died of tuberculosis. At the age of 12, the patient had inflammatory rheumatism and was confined to bed for about 6 weeks. There were no sequelae, cardiac or otherwise, following this disease. He had no history of diabetes, cancer, or tuberculosis. However, because his mother had been tuberculous, he was concerned about his susceptibility to this condition, and had had repeated chest films and diagnostic tests made for tuberculosis, all of which were negative. As a child he had a tonsillectomy, and a submucous resection had been done in 1945. Otherwise, the patient had enjoyed excellent health.

On examination, the patient had the general appearance of a man of 37 years. He was of average height and build and was not in acute distress. Only the left heel presented an orthopedic problem. There was an enlargement about the size of a half-dollar on the superior lateral aspect of the left heel just adjacent to the insertion of the Achilles tendon. The left calcaneous was about one fourth thicker than the opposite one. There was considerable tenderness throughout the calcaneus when direct pressure was applied; however, it was not grossly tender and palpation did not cause much discomfort. The entire tissue felt thick and slightly swollen; no lymphadenopathy was noted either in the immediate area or elsewhere in the body. The swollen area did not feel hot as would an acute infective process.

Anteroposterior and lateral x-rays of both heels which had been taken on February 9, 1953, were brought to the office by the patient. The most pertinent lateral views are reproduced in Figures 1 and 2.

These films revealed sclerotic changes in the left calcaneus with some roughening of the periosteal edges. There also was a heel spur present about the size of the fifth finger tip with three small areas of radiolucency which were about the size of a match-head. The osseous structure of the right calcaneus and foot appeared to be normal. Anteroposterior, lateral, and tangential x-rays were taken on November 23, 1953, and are reproduced in Figures 3 to 7.

In the x-rays taken on November 23, 1953, the right foot appeared to be normal from an osseus viewpoint. However, films of the left foot revealed considerably more sclerosis than the films of February 9, 1953. The heel spur was again noted. The three areas

Fig. 1

Fig. 2

Fig. 3

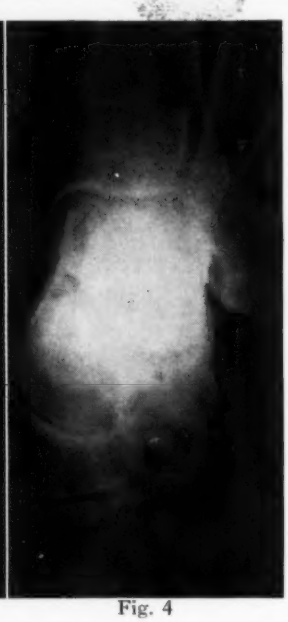

Fig. 4

of radiolucency noted on the previous films were absent. The tangential views not only clearly brought out the intense sclerosis, but there also appeared to be a typical sunburst effect of the left calcaneus with much roughening of its outer margin.

The diagnosis made at that time was (1) probable sclerosing type of osteogenic sarcoma or (2) possible sclerosing osteitis of Garré. Because of the possibility of the heel being sarcomatous in spite of the long duration of the condition, the patient was admitted to the hospital for a laboratory study and biopsy. The laboratory work and results were as follows:

Blood Count: November 27, 1953	
Erythrocytes	4,390,000 per cu. mm.
Hemoglobin	14.8 gms. per 100 cc. (94 per cent)
Color index	1.04
Leukocytes	9,650 per cu. mm.
Total polymorphonuclears	61 per cent
Segmented cells	58 per cent
Stab cells	3 per cent
Lymphocytes	32 per cent
Monocytes	5 per cent
Eosinophils	2 per cent
Sedimentation rate	16 mm.

Spinal Fluid Analysis: November 30, 1953	
Amount	10 cc.
Appearance	Clear
Leukocytes	None
Erythrocytes	None
Proteins	34.0 mg. per 100 cc.
Colloidal gold	1-2-3-3-4-3-3-2-1-1
Kolmer test	Negative
Clotting time	10 min., 10 sec.
Bleeding time	1 min., 27 sec.
Blood Chemistry:	
Proteins	5.5 grams per 100 cc. (normal 5.8-8.4)
Albumin	3.8 grams per 100 cc. (normal 3.6-5.6)
Globulin	1.7 grams per 100 cc. (normal 1.5-3.4)
Calcium	10.0 mg. per 100 cc. (normal 9.0-11.5)
Phosphorus	4.2 mg. per 100 cc. (normal 3.0-4.5)
Alkaline phosphatase	1.0 mm. units (normal 0.8-2.3)
Acid phosphatase	0.38 mm. units (normal 0.13-0.63)
Serology:	
Kahn test	Negative
Mazzini test	Negative

Urinalysis: November 27, 1953	
Color	Yellow
Character	Slightly cloudy
Reaction	pH 7.5
Specific gravity	1.012
Albumin	Negative
Sugar	Negative
Acetone	Negative
Diacetic acid	Negative
Bile	Negative
Leukocytes	None
Erythrocytes	None
Epithelial cells	None
Casts	None
Bacteria	None
Crystals	Amorphous phosphatase

The findings on the chest films taken at this time were reported as normal. Of all the laboratory work done, the only two findings of note were the slight elevation in the number of leukocytes and the slight increase in the sedimentation rate. The result of the colloidal gold test, in view of the negative Kahn, Kolmer, and Mazzini tests, was apparently meaningless. It was interesting to note that the alkaline phosphatase was well within normal limits.

On November 28, 1953, a biopsy of the left calcaneus was performed. Non-touch skin technic was used throughout, and a 2-inch longitudinal incision was made lateral to the insertion of the Achilles tendon. Material was removed from the soft tissues of the area, plus a plug of bone throughout the transverse part of the calcaneus. Grossly the calcaneus was noted to be

Fig. 5

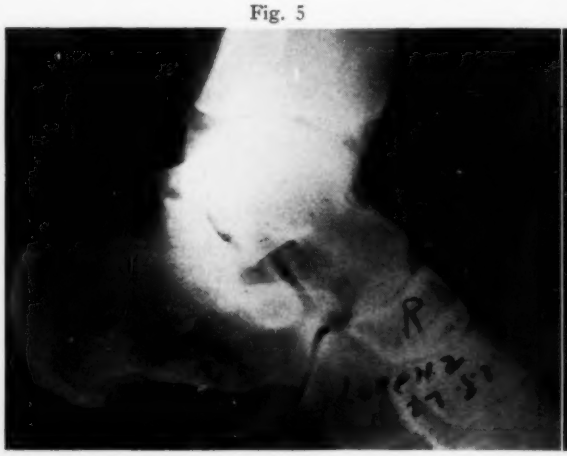

Fig. 6

Fig. 7

extremely sclerotic, even the central portion, and the entire bone was quite thickened. I felt so sure that this was neoplastic that I did not bother to take cultures as I should have done at that time. The patient's stay in the hospital was uneventful. No drainage occurred and there was no pain in the heel other than the usual postoperative incisional soreness. The patient was placed on antibiotics prophylactically for a few days following surgery. Reproductions of the microscopic visualizations are shown in Figures 8 to 11.

The pathologist reported that the paraffin sections of decalcified bone revealed many ragged surfaces of the bony trabecular areas with irregular cement lines and a mild accumulation of plasma cells and lymphocytes. He noted that these were seen in greater numbers where the fibroblastic proliferation within the haversian canals was relatively increased. In one section which incorporated fibrotendinous tissue attached to the bone, a single aggregate of lymphocytes and plasma cells was observed. No parts of the sections revealed evidence of tumor, and no tubercles or granulomatous areas were found. There was no evidence of suppuration. The pathologist's diagnosis was nonsuppurative chronic osteomyelitis.

The patient's postoperative progress was uneventful. He continued to have little or no pain following biopsy and was discharged from the hospital on his sixth postoperative day. Antibiotic therapy, sulfathiazole, 15 grains every 4 hours, was continued after his discharge. The patient had received penicillin and streptomycin in combination while hospitalized. He was advised to use a cane or crutches and, following his discharge, was seen periodically at the office.

In view of the pathologist's report and the absence of neoplastic disease, attention was then directed to the heel spur noted on the x-rays. This continued to give the patient difficulty when direct pressure was applied to the area, but the throbbing type of pain he had prior to biopsy no longer occurred. In the beginning, treatment of the calcaneal spurs was conservative, with bilateral use of Thomas heels with a 3/16 inch medial lift.

On February 5, 1954, the patient again had some throbbing pain and was placed on sulfathiazole, 15 grains four times a day. He obtained marked relief after several days. The total course of sulfathiazole amounted to 107½ grains at that time.

Radiographic examination on February 23, 1954, revealed less sclerosis and some retrabeculation. The patient had returned to work by this time and was progressing most satisfactorily except for the typical symptoms of the heel spur which were not relieved by the usual methods of wedges, sponge rubber pads, and the like. Therefore, on May 1, 1954, operation was performed to remove the heel spur.

Non-touch skin technic was used. A 2-inch medial incision was made longitudinally, and a spur the size of the fifth fingertip was removed from the left calcaneus. Fascia was stripped around the calcaneal area, and the area was curetted quite thoroughly. Sulfa was frosted into the wound, and a primary closure was made. The bone spur that was removed was sent to the pathologist. His report follows:

Clinical Note.—37-year old male.

Gross Findings.—The specimen consists of approximately 2 tablespoonfuls of bone fragments, some of which show adherent fibrotendinous tissue. The fragments measure from 10x5x5 mm. to 20x10x10 mm. in greatest dimensions. They are decalcified for sectioning.

Microscopic Findings.—Paraffin sections show fragments of irregular trabecular bone accompanied by pieces of periosteum. In most instances the fatty marrow displays a sprinkling of plasma cells, lymphocytes, and occasional eosinophilic polymorphs, and in one fragment especially these cells are encountered in greater numbers. Often they congregate in perivascular locations, but in other areas have infiltrated diffusely amid a rather scanty fibroblastic connective tissue. The bone trabeculae, especially in the latter instance, show uneven blue cement lines. No criteria of malignancy are observed. Although the changes are not as striking as the characteristic textbook picture, they do fit the necessary criteria of sclerosing osteitis of Garré.

The second surgical wound also healed without drainage, and the patient came to the office for follow-up care. He returned to work on May 17, 1954 and has had no pain or difficulty since that time.

Fig. 8

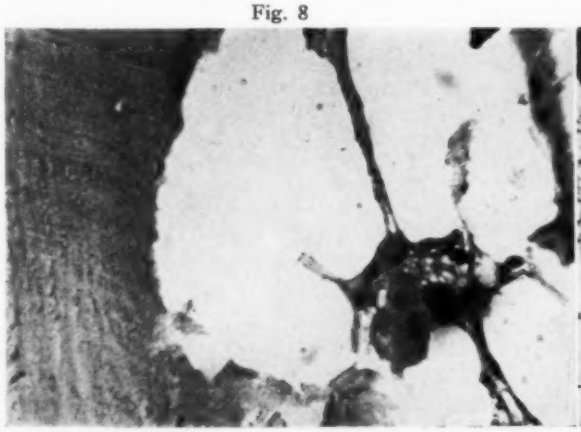

Fig. 9

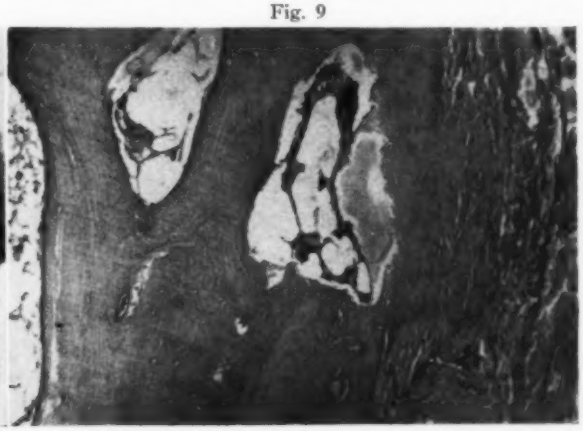

Fig. 10

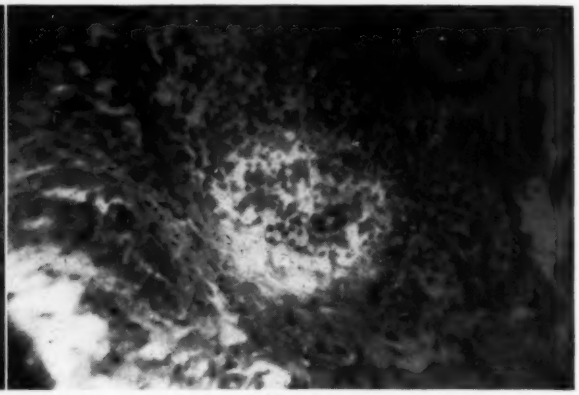

Fig. 11

Films taken on March 20, 1954, are shown in Figures 12 and 13 for comparison. The last x-rays, taken on June 1, 1954, reveal a rather marked improvement over the previous films and are reproduced in Figures 14 and 15. It can be noted that the hole persists where the tissue for biopsy was taken; however, considerable decalcification throughout the entire calcaneus can be seen on these films. The calcaneus has lost its original sunburst effect, and, of course, the heel spur has been removed.

DISCUSSION

A search of the literature reveals that very little has been written about sclerosing osteitis of Garré. Bick[1] states:

> In 1891, Garré described a type of chronic osteomyelitis in which the bone and periosteum became irregularly hypertrophied, but which never formed actual pus. To this lesion he gave the name sclerosing non-suppurative osteomyelitis. A special type of infection of bone was that first recognized by Ollier in 1864 and reported ten years later by his pupil, Poncet, who called it periostitis albuminosa. He believed it to be a local cortical and subcortical involvement by organisms of low virulence. Kuth described it in greater detail in 1938, and was able to trace sixty-six cases reported up to 1934.

Luck,[2] in his textbook, "Bone and Joint Disease," fills about one page discussing all types of nonsuppurative chronic osteomyelitis, including the following: (1) sclerosing osteomyelitis, (2) fibrous osteomyelitis, (3) osteomyelitis variolosa, (4) typhoid osteomyelitis, and (5) undulant fever osteomyelitis.

Howorth,[3] in his "Textbook on Orthopedics," discusses sclerosing osteitis of Garré in about a half page.

Steindler,[4] in his "Post-Graduate Lectures on Orthopedic Diagnoses and Indications," devotes a paragraph to the nonsuppurative type of pyogenic osteomyelitis. However, he states that it was first reported by Garré in 1893 rather than in 1891.

Steindler also points out that this condition is commonly considered to be neoplastic rather than infective because of the absence of destructive lesions. However, most physicians agree that because of the demonstrable inflammatory elements it is and should be classified as osteomyelitis.

The authors agree that the symptoms are relatively mild, chronic, and localized to the affected bone. The persistent and somewhat throbbing pain is the characteristic symptom. The most common clinical finding is enlargement of the bone with mild inflammatory changes. A slight leukocytosis and the elevation of the sedimentation rate are generally agreed to be the only pertinent laboratory findings. Characteristic features of this disease are seen roentgenologically. These have been well described.

Murray[5] discussed tender heel due to Paget's disease. Paget's disease should be mentioned as a possibility in differential diagnosis; however, after reviewing the x-rays, the case which I have described was certainly more easily confused with neoplasm, especially the sclerosing type of osteogenic sarcoma. It certainly was not one of Paget's disease.

SUMMARY

1. A case of sclerosing osteitis of Garré has been described.

2. There has been little written about this entity, and its etiology is somewhat controversial.

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Fig. 12

Fig. 13

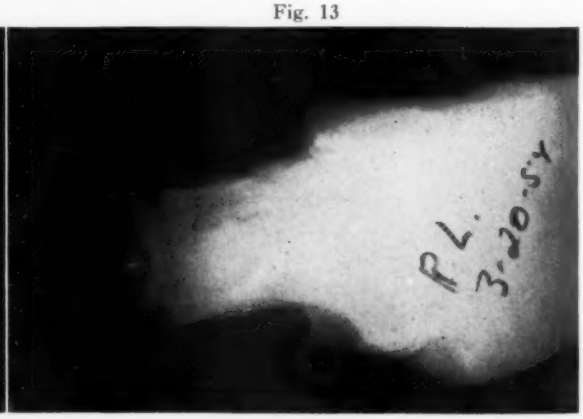

Fig. 14

Fig. 15

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A Commentary on Spinal Puncture, Including Myelography and Diskography

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Man's back has for many years been the subject of investigative research and will undoubtedly continue to present problems of diagnostic significance as long as man continues to walk on his hind limbs. Since there is no immediate prospect of man's changing his mode of locomotion, he will continually be confronted with stresses and strains of weight-bearing in the vertical position. If the automobile continues to displace the legs as man's chief means of propulsion, his low-back ills will undoubtedly become increasingly important causes of disability and ill health.

In 1874 Andrew Taylor Still established a new school of medicine, which he called osteopathy, and in so doing enunciated certain principles about the causes of diseases and about their treatment by manipulative procedures directed at the spinal segments. Still's theories, which have long since been proved clinically, have had a profound influence on the thinking of investigators doing research on diseases of the spine.

The field of vertebral mechanics has occupied the minds of men since the establishment of the osteopathic school of practice, and has, undoubtedly, led to research in this field by others. Today even the combined findings of all persons who have studied this field do not clearly reveal the true story of the many, varied diseases influencing the human back.

In 1891 Quincke[1] introduced the lumbar puncture as a means of obtaining cerebrospinal fluid for diagnostic study. Since that time, we have learned much about this fluid. During the first two decades after Quincke's discovery, the study of the cerebrospinal fluid was almost entirely confined to estimating the cellular content, to rough qualitative evaluations of the protein content, and to such special technics as the Wassermann and colloidal gold tests. Mestrezat,[2] in 1912, opened a new era by showing that chemical analyses of the cerebrospinal fluid offered diagnostic aids comparable to those obtained by chemical examination of the blood. Since then, many investigators have had the opportunity thoroughly to investigate the cerebrospinal fluid under conditions which permitted evaluation of fluid taken from medical, surgical, and neurologic patients in quantities large enough to establish standards, which are of diagnostic significance in any complete examination of the fluid.

Between the time of Quincke's original discovery —that cerebrospinal fluid could be obtained by inserting a needle into the subarachnoid space—and today, we have used this method not only for extracting fluid but for introducing anesthetics, medication, and opaque agents for the study of space-occupying lesions of the spinal canal. Since we, as orthopedic surgeons, are greatly concerned with the invasion of the subarachnoid space to confirm or rule out the possibility of an intraspinal lesion, we are also necessarily concerned with the technic of inserting the needle so that no actual harm can result from the insertion. We have all heard for years from patients who complain of a constant backache, dating from spinal anesthesia given for an appendectomy several years earlier and who blame spinal anesthesia for their low backache. Were this complaint comparatively rare, we might attribute it to the psychologic effect of having a needle inserted into one's spine. But since it is relatively common among patients who have had spinal anesthesia, it would be well to review and reconsider the possibilities of actual harm, however temporary, that result from the insertion of a spinal needle, whatever the purpose.

Some physicians are adept with the hypodermic needle and can insert one into a vein or into the subarachnoid space with utmost ease and without any trauma. And then, we have all also seen physicians who, in spite of much experience, will never learn to insert a needle successfully and without trauma into

either the spine or a blood vessel. So it becomes a matter not only of personal training and skillful adaptation of a correct technic, but also an appreciation for structures through which the needle must pass, and the trauma which may ensue if the needle is inadvertently positioned incorrectly.

For a diagnostic lumbar puncture, it is well to use the smallest needle compatible with the amount of fluid to be withdrawn and to precede the puncture with intradermal anesthesia to produce a wheal. Number 22 or 23 needles will cause little injury when inserted under anesthesia, with the patient in moderate flexion, directly through the supraspinous, the interspinous, and the flaval ligaments, through the dural sac, and into the subarachnoid space. It can be done if the patient is relaxed and is not hurt by the insertion of the needle. If x-ray pictures of the patient's back are available, it is well to study the anteroposterior and lateral views of the lumbar spine to determine what might complicate insertion of the needle. This step could often save the physician needless embarrassment and the patient needless discomfort. We know that spinal canals vary in diameter, and it is well to take into consideration the configuration of the spinal canal before entering it with a needle. We see the long, narrow, comparatively deep thecal sac or the wide, broad comparatively shallow thecal sac in other physical types in roentgen visualization with an opaque agent. It is certainly advisable to ascertain the type of sac by means of x-ray before inserting the needle.

Proper insertion of the needle will depend on the bony and ligamentous structures which the needle must penetrate and should aim for the center of the spinal sac. Among the factors which may alter the direction of the needle are the patient's position on the table and the curve and rotation of the lumbar spine, and the tight spine with spinous processes that actually kiss. In such cases, it may be advisable to insert the needle from a position lateral to, and on an angle toward the center of, the spinal canal. The size of the spinal canal will again determine the accuracy of such an insertion.

The thickness of the dura and the amount of tension produced by the fluid within the thecal sac will determine what sensation the tip of the needle will produce on passing through the dura and the arachnoid membrane. Again, the result depends on individual variations of pressure and the size of the thecal sac. Patients with comparatively low pressure within the thecal sac have no sensation when the needle passes through the dura and the arachnoid membrane. Those with comparatively high pressure feel a distinct pop when the needle passes through the sac.

The size of the needle will also determine what the patient feels when the needle penetrates the dura. Certainly a 25 gauge needle will meet with less resistance than an 18, 20, or 22 gauge. The small needles are in common use today and certainly minimize the trauma, as compared to the larger needles. It may be advisable to use a short cannula to facilitate the passage of a 25 gauge needle through the interspinous ligament, for the trauma resulting from the use of such a cannula would be minimal and would in no way affect the neural canal. With such a needle, the withdrawal of fluid is extremely slow, but, with patience, one can accumulate enough cerebrospinal fluid for the average examination. To introduce anesthetic solutions into the spinal canal, a 25 gauge needle is preferred. Again, the technic of insertion changes as the diameter of the needle diminishes.

The technic of administering spinal anesthesia has improved so much that serious complications are unusual. In the past 13 years at Detroit Osteopathic Hospital, spinal anesthetics have been administered to more than 39,000 patients, with no serious reactions. Placement of the needle into the spine has, however, in each case been under the direct supervision of a highly trained anesthesiologist. The head of the Department of Anesthesiology, Dr. Maurice Howlett, informed me that hospital records disclose only one case in which nerve injury could be attributed to the anesthetic solution or the needle. The injury manifested itself by toe drop and by muscular weakness and pain in one leg. The neurologic defect cleared up in approximately 2 months, and recovery was complete. Dr. M. L. Axelrod cites more than 27,000 cases of subarachnoid block with no serious complications. It has become increasingly evident that, with proper technics, a needle can be inserted into the subarachnoid space with little risk of trauma either to the arachnoid membrane or to nerve filament.

Since many orthopedic surgeons and neurosurgeons enter the subarachnoid space to introduce opaque agents into the lower lumbar spinal sac to obtain diagnostic visualization, our interest centers on how they perform this procedure. Too often, a patient who has had myelography done elsewhere complains of the pain produced by the procedure. Dandy[3] once told of the "terrible ordeal" endured by the patient who undergoes myelography. The pain need not, and should not, occur; nonetheless, in inexperienced hands it can occur, and then myelography gets the bad name. Certainly, one not adept and not adequately trained to place a spinal needle should not attempt myelography or even to withdraw cerebrospinal fluid. The complications of myelography can be minimized to the same extent as in anesthesiology, if the prime requisites are observed.

There is no substitute for clinical diagnosis, and myelography was never intended as a prime diagnostic technic. With this thought in mind fewer myelograms will probably be made. The percentage of myelograms yielding negative information increases in direct proportion to the histories and physical examinations that are inadequate. Even the size of space-occupying lesions of the spinal canal can be estimated with fair precision on the basis of physical diagnosis alone. Myelography should be reserved to confirm the clinical evaluation.

When Pantopaque is used for roentgen visualization of the subarachnoid space, an 18 or 20 gauge needle is proper. When Lipiodol was used routinely, the 18 gauge needle was almost mandatory, although in hundreds of cases a 20 gauge needle was used. Time is the only factor of consequence involved in using the smaller needle. The recovery of Lipiodol from the spinal canal with a needle finer than an 18 gauge is extremely slow. Even with Pantopaque, which has a viscosity much lower than that of Lipiodol, the recovery is delayed with 20 or 21 gauge needles.

The potential trauma from an 18 gauge needle must be weighed against potential irritation from the subarachnoid injection of the opaque agent. Since the opaque agent is withdrawn shortly after its administration, usually within 30 minutes of the injection, it would seem illogical for irritative reactions to arise in so short a time. However, we do see patients who are sensitive to the opaque agents being used today and who have allergic reactions and temporary arachnoiditis even though the oil remains in the spinal canal only for

a short time. On the other hand, we also see delayed healing of puncture wounds made by 18 gauge needles —probably more often than if a smaller needle were used. The trauma induced by the larger needle, as it penetrates the dura and arachnoid, is potentially greater than the danger of an allergic reaction and, in most instances, should be avoided.

Certain fundamental rules should be followed in performing myelography. I am sure it would not be out of place for me to review again the technic I employ.

The patient is given medication as though for an ordinary surgical procedure and placed in the prone position on the fluoroscopy table, usually with a small pillow under the abdomen to flatten out the lumbar curve. I have always found it advisable to anesthetize the skin and superficial tissues, especially the interspinous ligament, before I insert the needle. If the local anesthesia is adequate, few patients will complain about painful needle insertion. The prone position is comfortable for the patient and more relaxed than being upright or lying on his side. It is no more difficult to insert a needle into the spine in this position than when the patient is in acute flexion on his side. Certainly, it is much less troublesome for the patient. We must be on the alert to keep dull needles off our work trays, since they may induce trauma to the arachnoid membrane and dura, with an undesirable residual defect. I have found it wise to use short-bevel needles both for injecting and extracting oil. These, I believe, minimize the tendency toward trauma and diminish the surface against which a membrane or nerve filaments may be drawn during oil removal. Two-hole needles are to be avoided, since they provide no distinct advantage over conventional needles. I have even seen complications arising from the withdrawal of oil from the subarachnoid space: A piece of membrane incarcerated in the side hole as a result of the suction applied during the withdrawal, or a piece of membrane caught and actually torn from within the subarachnoid space when the stylet was replaced.

The ideal needle for myelography with an opaque oil is a 20 gauge short-bevel needle. If all of the principles of needle insertion are followed, such a needle is relatively nontraumatic and permits rapid and complete extraction of the oil. No needle larger than an 18 gauge should ever be placed within the subarachnoid space.

It is most important for the needle to be inserted as near the midline of the spinal sac as possible. Potential trauma to the caudal fibers or to the nerve roots at their emergence is an ever-present danger whenever the needle enters the lateral aspect of the spinal sac, regardless of the side. If one is not sure of the location of the point of the needle, he should seek the assistance of the radiologist to locate the point in relation to the interpediculate space. If the needle is too far to one side, or more than 3 mm. from the midline, it should be withdrawn and reintroduced. However, in reintroducing a needle, we add the problem of a second puncture of the dura and the arachnoid membrane. It therefore behooves us to develop a technic of insertion that will avoid this complication.

I would also consider the extradural or subdural injection of opaque oil in myelography as a complication. It is well to confirm the subarachnoid injection of the oil after 0.5 cc. has been injected, to make absolutely sure that the oil is within the confines of the subarachnoid space. Incomplete withdrawal of the oil would also be considered a complication. Only experience in withdrawing oil can teach when the evacuation is proceeding successfully. Cooperation with the radiologist is essential at this time, for it is necessary to keep the oil in a pool under the point of the needle if we are to evacuate the subarachnoid space completely. If more than one or two droplets remain within the confines of the subarachnoid space, I would consider it a complication. Although most authorities agree that Pantopaque is absorbable, there probably is also no question but that it does produce a degree of arachnoiditis which, in turn, produces a reaction that causes a defect in that membrane. For that reason, the oil should in every case be completely withdrawn within a reasonable time—that is, within 1 hour after the injection. If this were always done, we would have no reason to expect neurologic defects following the procedure.

Lindblom,[4] Cloward and Buzard,[5] Eaton,[6] and others[7,8] have advocated the use of a water-soluble opaque agent for injection directly into the nucleus pulposus of the disk as a means of determining the adequacy of the annular structures of the disk and their ability to maintain the nucleus within its normal confines. I believe, however, that most men experienced in the field have avoided this procedure as one that is both potentially dangerous and unnecessary. It requires puncturing both the posterior and the anterior dura and the arachnoid membrane and inserting the needle directly through the annular fibers into the nucleus pulposus. One eminent neurosurgeon[9] feels that conventional methods of diagnosis will yield the same results without the potential harm. A needle cannot be inserted directly through the annulus and into the nucleus without some damage, however slight, to the annular fibers and, what is even more important, possibly to the cartilaginous plate, particularly if the needle is inserted at a slight angle to the intervertebral space. It has been conclusively established that the loss of nuclear fluid—in other words, degenerative dehydration —takes place through pathologic rents or cracks in the cartilaginous plates. As a consequence, insertion of a needle through a plate would produce an initial rent, which could well lead to later dehydration and degenerative disease of the disk. I also feel that a needle large and rigid enough to be inserted through annular fibers into the nucleus will also serve to produce in these fibers some destructive change, which may become a site for ultimate rupture.

If clinical diagnosis reveals a potential disk rupture at the fifth lumbar vertebra, for example, and a needle is inserted into a diseased disk, no further deterioration could result from placing a needle into this type of disk. However, routine insertion of needles into the last three disks of the lumbar spine does not appear at all selective as a specific aid in searching for the diseased disk. The potential harm seems to me too great to warrant routine injections of this type when more conventional and less traumatizing avenues exist for achieving essentially the same result. This procedure may still be too new for a concise evaluation of the potential danger, and it may take several years before there is clearer knowledge of this problem. I am sure that the men who are working in this field are keeping accurate records. It will be interesting, in years to come, to see what happens to normal disks that have been invaded by needle, for injecting an aqueous opaque agent.

SUMMARY

I have attempted to call your attention to some of the potential dangers involved in spinal puncture, especially when the needle is inserted for the diagnosis of space-occupying lesions of the spinal canal.

To summarize briefly the principles that will insure nontraumatic entry into the subarachnoid space, without subsequent neurologic defect, let me say that no substitute can replace skill, experience, and accurate clinical diagnosis. The needle used should be sharp and short-beveled. It should be inserted with the bevel parallel to the long axis of the thecal sac. It should always be inserted near the center of the sac and should be as fine as is compatible with the procedure to be carried out. For Pantopaque myelography, the needle of preference is the 20 gauge. It is left in place throughout radiography, so that withdrawal of the oil may be expedited and only one puncture be made. Two-hole needles have no demonstrable advantage over conventional ones and, as a consequence, are to be avoided. The actual injuries this type of needle can produce have been mentioned. To insert a needle directly through both dural surfaces, through the posterior longitudinal ligament, through the annulus fibrosus, and into the nucleus pulposus so that a radiopaque solution can be injected for evaluating the adequacy of the annulus is unnecessary and fraught with danger. For conventional myelography, the needle should be inserted under local anesthesia with the patient prone and relaxed. The oil should be completely removed after radiography.

If all of the foregoing principles are followed, inserting a needle into the subarachnoid space and conventional myelography become compatible with excellent surgical care, with happy consequences for the patient.

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Benign Bone Cyst
Part I. Study and Case History

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Bone cyst was first recognized by Virchow[1] who in 1878 described it, after finding one in the humerus at autopsy. Heineke,[2] in 1903, was the first to study this lesion by means of a roentgenogram. The classification by Bloodgood[3] in 1910 of cystic conditions occurring in bone has been followed by most subsequent writers.

ETIOLOGY

To date, no agreement has been reached on the etiology of the bone cyst. Several theories have been advanced. One of these traces the origin to trauma with the development of hematoma. Thiemke,[4] Lang,[5] and Jenckel[6] attempted to prove that hemorrhage into the bone, in which the blood is for some reason not absorbed and organized, causes cyst formation. Pommer,[7] in his theory of intramedullary hemorrhage, assumed that a solitary bone cyst results from the encapsulation and alteration of a focus of intramedullary hemorrhage that fails to undergo resorption. Another theory, based on faulty calcium metabolism, attempts on the basis of malacia to show a relationship between bone cyst and Paget's[8] disease in adults or rickets in children. Another theory refers to infection. Pfeiffer,[9] after investigating and studying four cases, concluded that the circumscribed bone cyst is nothing but a softening center of inflammatory tissue in the bone marrow. Phemister and Gordon[10] contended that the solitary bone cyst represents the healed end-stage of localized, low-grade pyogenic osteomyelitis. Geschickter and Copeland[11] have advanced the theory that bone cyst and giant cell tumor result from abnormal hyperplasia of osteoclasts. Other theories trace the origin of bone cysts to bone infarct and to local post-traumatic dystrophy. Jaffe and Lichtenstein[12] explain both the solitary bone cyst and fibrous dysplasia as local defects in bone growth and development. All of these theories have had to yield to exception too often to be conclusive, and it must therefore be admitted that none, so far, is entirely convincing.

ONSET AND AGE OF INCIDENCE

The onset of this condition, which is a definite clinical entity with well-known signs, is usually during childhood or adolescence. It occurs more frequently among males, with the greatest incidence between the ages of 10 and 15. In the series of cases studied by James, Coley, and Higinbotham,[13] the average age of onset was 15.3 years. The youngest patient was 3 years of age and the oldest 45. Most of Jaffe's[14] nineteen patients were in the 3-to-14-year age bracket when first seen.

DISTRIBUTION

Usually, the long tubular bones are involved. The most common site of involvement is the humerus, then the femur, and finally the tibia. The cyst has a predilection for the metaphyseal region of long bones, particularly the proximal ends of the humerus, the femur, and the tibia. More rarely, the cyst may appear in the radius, the fibula, the ribs, the calcaneus, the phalanges, the pubis, and the ischium.

CLINICAL FEATURES

Pain is the most common complaint when there is a solitary bone cyst, but it also is often so mild or inter-

mittent that it fails to arouse the patient's suspicion. Often the pain is felt after strain or prolonged function. Or the first symptom may be a pathologic fracture, in which case a roentgenogram is diagnostic. Little if any swelling or discoloration is noticed in the area of cystic involvement.

PATHOLOGY

The gross pathologic appearance at the time of surgery is usually one of fusiform enlargement in the cyst area. The periosteum can be raised easily, and the underlying bone is thin and has a bluish discoloration. The cortex can be entered with ease, and the cavity is lined with variable amounts of connective tissue, ranging from a membrane that is obvious to one so fine that it may be difficult to see. The cavity contains a variable amount of fluid, usually yellow or brown, but it may be red if cyst exploration is done following trauma or a pathologic fracture. Ridges often extend along the cyst wall and only rarely is there partitioning into a multicameral or multilocular cavity. The lesion has been noted to involve the metaphysis and, when active, may even extend well into the diaphysis of the bone. It has never been found to involve the epiphyseal plate.

Microscopic examination offers little more information that is conclusive, and calcium and phosphorus metabolism and serum phosphatase levels are all within normal limits.

DIFFERENTIAL DIAGNOSIS

Although the solitary bone cyst is not difficult to diagnose, it must be differentiated from other lesions of the skeletal system. Some conditions to be considered in differential diagnosis are giant cell tumor, fibrous dysplasia, nonosteogenic fibroma, and osteitis fibrosa cystica (due to hyperparathyroidism). More rarely to be differentiated are neurofibroma, enchondroma, lipoma, and sarcoma.

The giant cell tumor begins in the epiphyseal end of the bone, reaching the shaft later, by progressive expansion. It usually occurs in the 20-to-40-year age group, with little difference in incidence between the sexes.

Although fibrous dysplasia is usually recognized in childhood or early adult life, it may occur at any age. Females are affected more often than males. In it, the long bones are usually involved, although the flat bones may sometimes be involved. The monostatic form, arising in a long bone of an extremity, resembles the solitary bone cyst. The serum phosphatase level may be elevated, and serum calcium values are normal. In gross pathologic appearance, the lesion bears little resemblance to the solitary bone cyst, for it consists of dense gray or white fibrous tissue, thinly or thickly trabeculated with poorly calcified fibrous bone.

Nonosteogenic fibroma generally occurs in older children and adolescents. The tumor is usually in the long bones and more often involves the bones of the lower extremity than those of the upper. The lesion is found in the upper or lower third of a long bone, at some distance from the adjacent epiphyseal plate.

Hyperparathyroidism usually involves young to middle-aged adults. In it, the blood chemistry is altered so that serum calcium and phosphatase values are elevated and the serum phosphorus level is diminished. In its early stages, it frequently involves decalcification, which may later be associated with cysts or tumors. The bone lesion is generalized, usually occurring in the diaphysis of the bone.

Fig. 1. Fracture of the distal right humerus in the area of cystic degeneration. Anteroposterior and lateral views of arm in cast, October, 1948.

TREATMENT

Three basic forms of treatment have been used: roentgen therapy, resection of the cyst, and bone grafts. Any number of combinations of these treatment methods have been used. Surgical treatment, combining curettage with bone grafting, has consistently given the most satisfactory results. Roentgen therapy may result in retardation of bone growth by damaging the epiphysis, secondary malignant lesions, or failure to obliterate the cyst. Garceau and Gregory[15] have demonstrated that, regardless of the type of treatment given, the cyst in its active, early stage is likely to recur after treatment, whereas in its late, latent stage it responds satisfactorily. If the lesion, when first seen, is associated with a fracture, the initial treatment should consist of routine fracture care. This does not mean disregarding surgical treatment for the bone cyst, but the fracture care is an emergency procedure, whereas the care of the cyst is elective. Moreover, it is well to remember that the cyst may be obliterated following the fracture.

CASE REPORT

A girl, aged 14, was hospitalized in April, 1954, complaining of a continuous pain in the right elbow. She had had progressive, recurrent pain in this area for several months whenever she used the arm exces-

Fig. 2. Anteroposterior and lateral views of healed fracture area out of cast, showing that cyst area was only partially obliterated by callous formation.

Fig. 3. Anteroposterior and lateral views of cystic degeneration of the right humerus as seen when patient was admitted to hospital in April, 1954.

Fig. 4. In July, 1954, 3 months after surgery, x-rays showed evidence of bone regeneration and obliteration of the cyst area.

sively. Her history revealed that in 1948, after a fall, the distal third of the right humerus had been fractured through an area of cystic degeneration (Fig. 1). After routine fracture care, she had had an uneventful recovery (Fig. 2).

At examination upon admission to the hospital, the distal portion of the right humerus appeared slightly swollen. Motion in any direction or palpation caused pain. Roentgenograms revealed an expansile destructive defect in the lower half of the distal right humerus with some extension into the upper humerus (Fig. 3). Destruction of the medullary bone and of the bone on the inner side of the cortex was also evident. Roentgenograms of other long bones and the pelvis, spine, and skull disclosed no pathologic lesions. The results of the urinalysis and complete blood count and the serum values of calcium, phosphorus, and phosphatase were all within normal limits. Tests for venereal disease gave negative results. Sedimentation rate was 37 mm. per hour.

An operation, performed on April 28, 1954, consisted of curettage of the cyst and implantation of cancellous bone substance from the crest of the ilium. At the time of surgical exposure it was noted that the bone was thin and had a bluish discoloration. A window, cut through the cortex, disclosed that the cavity was lined with a fine fibrous membrane and contained a large amount of reddish-brown fluid. After the operation, the arm was placed in a protective plaster splint. The independent reports of two different pathologists contained a diagnosis of benign bone cyst. Roentgenograms taken in July, 1954, showed definite evidence of bone regeneration and obliteration of the cyst area (Fig. 4). When last seen in October, the patient was symptom-free and had full functional use of the right arm, which was solid to palpation.

SUMMARY

Bone cyst has been discussed. An illustrative case, involving a young girl, has been presented. It has been pointed out that although roentgenograms are extremely helpful in identifying the bone cyst, positive differentiation from other bone diseases is not always possible. The differential factors in accurate diagnosis have been stated, and most successful therapy has been discussed.

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Part II. Radiographic Differentiation

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The case described by Dr. Fridena in the preceding article is interesting from the standpoint of differential diagnosis, for certain features of the bony lesions that involve the metaphysis and diaphysis of the lower humerus are not entirely typical of simple benign bone cysts.

Two independent histopathologic reports on the surgically extirpated tissue classified the lesion as benign bone cyst, yet both manifested considerable interest in the evaluation of the parathyroid glands, the radiographic appraisal of the rest of the skeletal system for possible cysts elsewhere, and the gross appearance of the lesion, as well as the radiologist's opinion of the lesion.

The purpose of this paper is to mention briefly certain other bone diseases that on radiographic study may, to greater or lesser degree, simulate benign bone cyst.

1. *Giant cell tumor,* although also an osteolytic lesion divided by trabeculation into irregular soap-bubble-like compartments, nevertheless involves the epiphysis, rather than the metaphysis and diaphysis. The youth of Dr. Fridena's patient and the absence of the marked expansile character of typical giant cell tumor help to exclude that entity.

2. *Medullary fibrosarcoma,* which is relatively rare, may resemble central chondroma or giant cell tumor.[1] Like the lesion in question, it occurs in the shafts of long bones near the epiphyseal line and produces an irregular expansion of the bone. It may occur at any age.

3. *Hyperparathyroidism* (Recklinghausen's disease or osteitis fibrosa cystica), when seen radiographically, presents a multilocular appearance. It involves the diaphysis and may result in a pathologic fracture like that described by Dr. Fridena. The low incidence of hyperparathyroidism in children of 14, the lack of generalized decalcification of the skeletal system, and the normal blood chemical values all help to exclude this possibility, however.

4. *Fibrous dysplasia,* whether monostotic or polyostotic, must always be considered, for it may occur at any age and involve any portion of a flat or long bone. In it, however, pathologic fracture is rare.[2,3]

Radiographically fibrous dysplasia may resemble simple bone cyst. However, a bone cyst usually involves the upper metaphyseal region of the humerus, tibia, or femur. The so-called cysts of fibrous dysplasia appear on roentgenograms as rarefied areas of bone. Histopathologic study shows that the areas are composed of solid islands of collagenous connective tissue or fibrous tissue, often distributed in whorls. Within these whorls innumerable superimposed, coarse, curving, "fuzzy" columns of bone may be seen. These various components impart the appearance of a multilocular lesion which obviously has greater density than one composed of fluid, as is the case in simple bone cyst.

Moreover, the increased density at the peripheries of the apparent "cysts" of fibrous dysplasia often extends for varying distances into the adjacent bone, shading off with a tonguelike configuration into that bone and imparting a characteristic "candle-flame" appearance to the lesion.[4] This did not apply in the case under discussion. True cysts in fibrous dysplasia result only from degeneration, necrosis, or hemorrhage.

5. *Enchondromas or chondromas* may closely simulate fibrous dysplasia or benign bone cysts. However, their margins are usually sharp with no alteration in the structure of the adjacent bone and none of the shading-off that is found in fibrous dysplasia. As has been mentioned, central chondroma is a circumscribed area of radiolucency near the end of a long bone that may be confused also with giant cell tumor, medullary fibrosarcoma, or nonosteogenic fibroma of bone. Since central chondroma occurs between the ages of 10 and 30, it might have been a consideration in Dr. Fridena's case if the lack of the multilocular character of chondroma had not ruled against it.

6. *Dyschondroplasia* (Ollier's disease) may present radiographic evidence of irregularly distributed rarefied areas in the metaphysis and diaphysis with some thinning of the cortex, but no periosteal reaction or new bone formation is noted.[5] Moreover, it is a familial disease also characterized by bowing of the long bones with distortion of the ends of those bones. This was not true of the case under discussion.

SUMMARY

Differential diagnosis of multilocular, rarefied, or osteolytic bone lesion of the diaphysis and metaphysis of the lower humerus has been discussed.

CONCLUSION

It was felt that benign bone cyst was the most likely diagnosis, although the lesion occurred in the lower end of the humerus and in a girl aged 14. Malignancy was excluded by the pathologists' reports, although pathologic fracture of the bone was obvious. It was concluded that repeated trauma and hemorrhages with marked fibrous tissue reaction had resulted in the development of a benign bone cyst.

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Ischemia Due to Skeletal Trauma

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Other than arterial diseases, thromboembolic phenomena, and extrinsic factors, such as tumors, conditions that cause ischemia of the extremity include arterial spasm, compression, and laceration following skeletal trauma. The vascular complications of skeletal trauma can be insidious in onset and, if not recognized and treated early, may lead to contracture deformities or gangrene. Paradoxically, treatment given for skeletal trauma may also produce contracture deformities or gangrene.

Considerable attention is devoted in current medical literature to the treatment of deformities of the extremity, caused by ischemia. However, little information is available on the preventive treatment of this crippling condition, indicating that this aspect remains relatively unexplored. A year's survey of references on this subject has revealed that Thomson and Mahoney[1] and Griffiths[2] have pioneered in studying the causes and preventive treatment of ischemic contracture. Their work seems thus far to be the most complete in the field.

Whenever skeletal trauma causes arterial injury it is also apt to induce marked vasospasm of the injured vessel. This vasospasm is in itself a protective mechanism of the body but in the traumatized extremity, it can result in ischemia of muscles distal to the vasospasm. If ischemia develops, there is grave danger that either ischemic contracture of varying intensity or irreversible gangrene will follow.[3] Vasospasm in one extremity may, by reflex action, produce ischemia in the opposite extremity; or it may spread upward to the renal arteries and thereby cause lower nephron nephrosis.[4]

The ischemic contracture of an extremity traumatized by fracture, dislocation, or contusion was, until recently, believed to be the consequence of venous obstruction. As late as 1938, Christopher[5] supported this view when he stated that ischemic contracture results from interference with the venous return of blood, thereby causing overengorgement of the muscle bundles. Another view was advanced by Volkmann[6] who described this contracture 60 years ago. He attributed it to arterial obstruction, and his belief has since been substantiated by the observations of Griffiths[2] and of Thomson and Mahoney.[1]

Arteries may be injured in several ways. They may be severed or lacerated by a displaced bone or bone fragment, or they may be compressed by a displaced bone, a subfascial hematoma, or a tight cast. They may also be injured by contusion due to trauma, as in mangle or wringer injuries that crush the capillary and lymphatic networks.

Traction, too, may injure arteries and induce vasospasm, according to Thomson and Mahoney.[1] One of their patients was treated for a fractured femur with a bilateral gallows-type traction; 9 days later, ischemic contracture of the uninjured leg was diagnosed. Histologic study of a biopsy specimen of muscle, after the onset of ischemic symptoms, disclosed the presence of all forms of degeneration of the muscle bundle. Marked congestion of the blood vessels, occlusion of the capillaries by erythrocytic packing, and some infiltration of the muscle bundles by polymorphonuclear leukocytes were found. Thomson and Mahoney[1] feel that muscle degeneration is a sequel to capillary stasis due to deficient arterial blood-flow in the muscles. They found that in cases of ischemic contracture the nuclei of the muscles were shrunken and pyknotic, and the capillaries occluded. In cases without contracture complications, the nuclei remained oval and intact, and the capillaries patent. Ischemic muscles were also found to have marked cross-striations, whereas normal muscles have prominent longitudinal striations. Thus microscopic examination of biopsy material can help in the prognosis in ischemia.

The vessels most commonly injured by skeletal trauma, with production of ischemia, are the ulnar, radial, brachial, axillary, subclavian, femoral, and popliteal arteries. The radial and ulnar arteries are jeopardized by fractures of the forearm and by mangle or wringer injuries; the brachial artery by supracondylar fractures of the humerus, elbow dislocations, and mangle or wringer injuries; the axillary artery by fractures of the humeral neck; the subclavian artery in fractures of the clavicle; the femoral artery in fractures of the femur; and the popliteal artery in high oblique fractures of the tibia and in "bumper" injuries without fractures.[1, 4]

Ischemia is seen most often in the flexor muscles of the forearm. This may be because so much attention has been focused on ischemic contracture of the forearm that its occurrence in other muscle groups has been overlooked. That ischemic contracture is not limited to the upper extremity is adequately supported by Thomson and Mahoney[1] who reported forty-two cases of ischemic contracture between 1931 and 1947, twenty-four involving the upper extremity and eighteen the lower extremity.

The four main symptoms of ischemia following skeletal trauma are pain, pallor, pulselessness, and paralysis.[1, 2]

Pain may not be present or it may range in gradation from slight to excruciating. In the leg, the pain is felt in the calf muscles and is accentuated by dorsiflexion of the foot. In the arm, the pain is experienced in the flexor group of muscles and may be intensified by extension of the hand.

Pallor is an important early sign of ischemia. The extremity may be waxen or may range in color from the cyanotic mottling of the early stages to the gangrenous black of the later stages. Or, the involved area may be patchy and erythematous, and the hand or foot dusky red.

Pulselessness is another early sign of ischemia. However, although the pulse may be palpable some time after the onset of pain and pallor, it may disappear suddenly. The pulses can usually be palpated easily in the upper extremity but those in the lower extremity are less easily discernible. However, in a small percentage of patients, it may be normal for the pulses of either extremity to be indiscernible.[7] If the diagnosis is questionable, an oscillometer is invaluable for detecting the pulse and determining the oscillometric index.

Paralysis is a late symptom. By the time paralysis takes place, it is usually too late to prevent contracture.

Both the motor and the sensory nerves are involved. In addition to paralysis of the ischemic extremity, the patient may experience some type of anesthesia, which may later be confused with nerve injury.

In addition to the main symptoms already described, there will be hypothermia of the affected part and probably also edema. If these symptoms are observed following a skeletal trauma, one may be sure that ischemia is present. What is more, unless it is treated promptly, one may also be sure it will progress to some degree of ischemic contracture.. This may range from segmental contracture to gangrene, depending on how much ischemia there is and how long it has been present.

Since any vascular complication should be regarded as an emergency, treatment should be initiated as soon as it is diagnosed. However, *what* treatment should be given will depend on the case in question. For instance, when an artery has been lacerated and a hematoma has formed, the artery should be repaired. If the vessel is not reparable, it should be ligated and resected, and an autogenous venous, or a homogenous arterial, graft should be performed if possible. On the other hand when an artery is compressed, the condition may be corrected by accurate reduction of the fracture or dislocation and by expression of subfascial hematomas. When ischemia results from vasospasm, the problem becomes more difficult. In the past, procrastination seems to have been the most widely used approach even when the complication was diagnosed early. And today, the condition often goes unrecognized until it is too late to prevent deformities or gangrene. It therefore seems that emphasis should properly be placed on the need to develop an awareness that ischemia may follow skeletal trauma. This is important, for with the early recognition of ischemia procedures to relieve the vasospasm can be instituted, thereby preventing irreversible tissue damage. Since shock is an ever-present possibility, it also must be combatted with appropriate measures.

Main attention must center on methods for relieving vasospasm. All of the leading authorities[1, 2] agree on this, just as they agree on the need for prompt removal of all external pressure on the involved extremity—tight bandages, casts, and traction included. In some instances, surgical exploration of the injured site is necessary. If, for example, a tight fascia covers an artery, splitting the fascia may relieve the spasm. If this procedure fails to obtain the desired result, the sympathetic innervation of the vessel may be interrupted locally by bathing the spastic vessel with procaine. This may suffice to relax the vessel temporarily, as may also a periarterial sympathectomy or the intravenous administration of papaverine. But if none of the foregoing procedures relieve the vasospasm, the sympathetic fibers should be blocked at the ganglionic level. In the lower extremity, such sympathetic denervation can be accomplished by paravertebral procaine block of the lumbar ganglions, by high spinal anesthesia, or by lumbar sympathectomy. In the upper extremity, sympathetic innervation can be interrupted by procainization of either the brachial plexus or the stellate ganglion or by means of cervicodorsal sympathectomy. The use of sympatholytic drugs either orally or parenterally is not recommended in such cases. They produce vasodilatation of the normal arteries throughout the body, with little or no effect on the involved extremity. However, in my own practice, in three cases of pregangrenous ischemia due to arteriosclerosis obliterans, I have had gratifying results with the intra-arterial use of Priscoline in the involved extremity. The same procedure might produce similar results in the vasospasm complicating skeletal trauma. At least it might be worth a trial.

The study of arterial vasospasm discloses many baffling problems. Some physicians have obtained good results with certain types of therapy, whereas other physicians report unfavorable results from the use of identical therapy.

The experience of Herrmann[8] with a series of patients who had ischemia after skeletal trauma convinced him that passive vascular exercise is the most effective method of stimulating collateral peripheral circulation.

Watson-Jones,[4] on the other hand, believes ischemia can be avoided by elevating the injured or fractured extremity for several days or weeks after skeletal trauma, thereby controlling edema. He also believes that an unpadded plaster cast should not be applied immediately after injury or operative reduction of a fracture. He emphasizes application of a woolen bandage and defers the use of an unpadded cast for several weeks.

Thomson and Mahoney[1] treated ischemia with spinal anesthesia in seven patients between 1947 and 1951. Six of them recovered completely without the development of contracture. In the seventh, the treatment failed.

Gardner[9] reports four cases of vasospasm and ischemia after injury. All four progressed to gangrene, requiring amputation. He treated the patients with sympathetic block, intravenous administration of papaverine, and surgical exposure of the vessel. He found his treatment wholly unsatisfactory. As a result of these failures, it is Gardner's opinion that the vascular spasm which follows injury may be the organism's reaction to shock, mediated by the endocrine system. Since local treatment has been unsatisfactory, he suggests that further study may have to be directed at general, rather than local, treatment.

The observations of Innerfield[10] tend to support Gardner's conclusion. Innerfield's research has had to do with the action of trypsin (Parenzyme) in the vascular field. He found that it lyses intracapillary and lymphatic thrombi and prevents the sludging of blood, thereby increasing the blood flow at the capillary level, facilitating lymphatic drainage, and reducing edema. It may be that by thus maintaining patent capillary and lymphatic systems in the muscles distal to the vasospastic artery, any ischemic changes may well be attenuated or prevented. However, further research must be done on these types of injuries in order to prove or disprove the efficacy of the drug.

The traumatized extremity should be spared further injury by chemical, thermal, or mechanical agents. It should not be elevated except for very short periods, to reduce edema. Its ideal resting position should be slightly below the heart level. Never should heat be applied to an ischemic extremity, for it increases tissue metabolism, speeding the assimilation of oxygen and engorging the muscles with blood. These factors will increase the danger of contracture and gangrene. Refrigeration is used only after irreversible tissue changes have taken place. In such cases, amputation is necessary as soon as the patient's condition warrants the operation.

The treatment of mangle and wringer injuries merits special mention. In such cases, the treatment

may be faulty because only bruising is apparent immediately after injury. It may take several hours before the vascular crushing and the effects of the burn, with the necrosis and sloughing so often seen in such cases, become apparent. Following such injuries, the extremity should be elevated, and cold packs applied. If no laceration is present, a pressure dressing of petrolatum gauze and a splint should be applied, and the area examined frequently. If severe edema or hematoma develops, an incision is made, and drainage instituted. Phalen[11] has found cervical sympathectomy beneficial in controlling the edema, whereas Posch[12] has succeeded in reducing edema with hyaluronidase. Again, perhaps Parenzyme might help to maintain the patency of capillary and lymphatic vessels at the site of the injury.

In conclusion, I want to stress three factors that are of prime importance if one is to have any hope of success in preventing contracture deformities or gangrene after skeletal trauma: (1) an awareness that ischemia may follow skeletal trauma, (2) early diagnosis of the ischemia, and (3) positive treatment with no procrastination after ischemia has been diagnosed.

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Tibiofibular Diastasis

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The ankle is a hinge-type diarthrosis (ginglymus) which is formed by the lower end of the tibia and its malleolus, the malleolus of the fibula, and the inferior transverse ligament, which together form a mortise for the reception of the upper convex surface of the talus and its medial and lateral facets. The necessary stability of the ankle joint is obtained by the downward projection of the malleoli which form the clasping surface of the mortise on each side of the talus and permit only a slight degree of lateral movement.

The inferior talofibular articulation is a syndesmosis-type of amphiarthrosis which has no movement, save elasticity, which is important in maintaining the stability of the ankle mortise in trauma.[1] The tibula and fibula are connected by a strong interosseous ligament, the inferior transverse ligament, and the anterior and posterior tibiofibular ligaments.

Diastasis is a form of dislocation in which there is a separation of two bones normally attached to each other without the existence of a true joint.[2] Thus, a tibiofibular diastasis results in lateral separation of the mortise, causing disabling instability, insecurity of the joint, and eventually traumatic arthritis. The rupture of the inferior transverse tibiofibular ligament allows for the diastasis and accompanies second degree abduction fracture-dislocations of the ankle, when the fibula is fractured above the tibiofibular joint.

The diagnosis of tibiofibular diastasis is usually afforded by determining the space between the talus and the base of the internal malleolus. X-rays should be made with the bones in the position of strain and can best be evaluated by the local infiltration of an anesthetic agent to overcome the pain and muscle spasm which accompany trauma. X-rays should be taken of any severely sprained ankle with the foot held in full inversion and in full eversion. If the gap between the talus and medial malleolus on the inner side of the joint is so great that it cannot be accounted for by displacement of the lateral malleolus, it indicates diastasis of the inferior tibiofibular joint, and proper treatment must be applied accordingly.[3]

The torn ligaments heal by the growth of new elastic connective tissue, and it takes time and functional stress to transform this tissue into a dense ligamentous structure which will not stretch under functional strain. Any stretching will widen the mortise and render the ankle unstable, which is the reason the tibia and fibula must be held tightly together for a protracted period, and the reason delayed and long-protected weight bearing is necessary.

TREATMENT

Treatment of tibiofibular diastasis consists of the following:

1. *Closed Method.*—While the malleoli are held in firm approximation by manual manipulation, a plaster-of-paris cast is applied to form a tight support. There must be no forced dorsiflexion because the anterior portion of the talus, being wider than the posterior portion, has a tendency to act as a fulcrum and widen the mortise joint. The plaster of paris should be extended to the midthigh and the knee maintained in a semiflexed position. The cast must be closely observed and kept snug. There must be no weight bearing on the affected extremity for approximately 8 weeks, and plaster-of-paris immobilization should be maintained for 12 to 16 weeks.

Recurrence of tibiofibular diastasis following closed reduction is common, owing to the inefficiency of external fixation. Detection of postreduction tibiofibular diastasis can be afforded by frequent postreduction x-rays. As soon as the condition is diagnosed, open operation is advocated.

2. *Open Method.*—Approximation of the tibiofibular syndesmosis can be afforded by insertion of a supramalleolar tibial bolt or screw. It is important that the fixation engage both tibial cortices, and at the time

of the operative procedure overcorrection must be avoided by moderate dorsiflexion so that the widest part of the talus is engaged in the mortise joint. The fixation apparatus should be inserted approximately 1½ inches above the ankle joint and directed from the fibula to the tibia in an anterior-oblique plane. It is important that the metallic fixation apparatus have deep threads for mechanical security. Following the operative procedure, a long leg cast must be worn for approximately 6 weeks, and a short leg walking caliper worn for approximately 2 to 4 weeks. It is important that weight bearing not be allowed for 8 weeks whether the reduction is by the closed or open method. It is advisable to remove the metal fixation apparatus after 8 to 10 weeks because of the minimal amount of subcutaneous tissue in the area of the ankle joint.

CASE REPORT

A 32-year-old man was admitted to the hospital on October 23, 1952, with the chief complaint of pain in the right ankle which had been present since September 16, 1952.

Onset and Course: On September 16, 1952, at approximately 11 p.m., the patient jumped from a guard rail at his place of employment, after which he experienced pain and swelling of the right ankle. The following day his family physician was consulted, and the patient was sent to a hospital for x-ray examination. The radiologist gave the family physician a negative report, and conservative management for a sprained ankle, consisting of elevation, adhesive strapping, no weight bearing, and analgesics, was employed.

After 2 weeks on crutches, gradual weight bearing was allowed, and 5 weeks after the injury, the patient was ready to return to his occupation as a mechanic, although there was pain and swelling which caused him to limp upon ambulation. The company doctor would not allow the patient to return to his duty until x-rays were made, after which the patient was sent back to his family physician for further care.

Re-evaluation by the radiologist who x-rayed the patient initially revealed marked separation of the ankle mortise in comparison to the initial film taken 5 weeks previously.

Orthopedic consultation was secured, at which time a provisional diagnosis was made of tibiofibular diastasis with partial subluxation of the talus. The patient was admitted to the hospital on October 23, 1952; operation was performed on October 24, 1952, and the patient was discharged on October 27, 1952.

History: The patient had had the usual childhood diseases. The family history was negative. Herniorrhaphy had been performed in 1943. The fourth finger of the left hand had been lost in 1941.

Physical Examination: Examination revealed a well-developed white male weighing 175 pounds and 5 feet 6 inches in height. His blood pressure was 154/110, and he was in no apparent distress.

Physical findings were essentially negative except for an enlarged right ankle that was tender to palpation and, particularly, dorsiflexion. The skin over the lower third of the leg, ankle, and foot glistened; and there was evidence of passive congestion (swelling) at this site. The deep tendon reflexes were physiologically normal.

Intern's Impression: Possible essential hypertension, and traumatic injury to right ankle were noted.

Laboratory Examination: Findings were as follows:

Erythrocytes	5,180,000 per cu. mm.
Leukocytes	8,600 per cu. mm.
Hemoglobin	105 per cent-16.8 grams per 100 cc.
Schilling differential count	normal
Kahn test	negative
Urinalysis	essentially negative

Roentgenologic Examination: On October 23, 1952, anteroposterior, oblique, and lateral views of the right ankle showed evidence of lateral subluxation of the talus in relationship to the tibia. There is also evidence of altered changes affecting the medial malleolus which we believe represent an avulsion type of fracture of that area.

Preoperative Diagnosis: Right tibiofibular diastasis with partial lateral subluxation of the talus.

Operative Procedure: Open reduction of diastasis of right ankle with insertion of a wood lag screw.

Postoperative Diagnosis: Right tibiofibular diastasis with partial lateral subluxation of the talus.

Postoperative Roentgenologic Examination: Examination shows evidence of a satisfactory reduction of the talus, there being only slight gaping between the medial margin of the talus and the medial malleolus. There is evidence of a screw through the distal end of the tibia and fibula for fixation purposes. Studies show evidence of satisfactory reduction of subluxation of the right talus.

The postoperative hospital stay was very uneventful. There was no evidence of postoperative complications or circulatory impairment due to the plaster-of-paris cast.

The plaster-of-paris cast was worn until December 14, 1952, with recheck x-rays being made periodically. A walking caliper was applied, and the patient was permitted weight bearing. The external support was removed on January 10, 1953, and the ankle was protected with only an elastic bandage for support.

The patient was readmitted to the hospital on May 11, 1953, and on May 12, 1953, the screw was removed. There was no discomfort or disability due to the metal screw in situ, but there was an obvious bump on the lower leg, most noticeable on the medial surface. The patient acknowledged bumping the leg upon occasions caused soreness and pain for a few days; otherwise there was no disturbance.

On June 19, 1953, the patient was re-examined and x-rayed. He was found asymptomatic, and no evidence of widening of the mortise joint was present.

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Arthrogryposis Complicated with Secondary Dislocations

A Case History

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INTRODUCTION

Calling the staff together is the usual procedure when an unusual case is considered at the Los Angeles County Osteopathic Hospital. Thus, when an apparent dislocation of the right knee in a day-old infant resisted all efforts at closed reduction, I presented the case to the attending staff of the orthopedic department who made the diagnosis of arthrogryposis multiplex congenita with secondary dislocation of the right knee.

The term "arthrogryposis multiplex congenita" designates a symptom complex which includes congenital joint rigidities of various degrees. This term was initiated by Rosenkrantz in 1905 who collected fifty-six similar cases in the literature.[1] Stern[2] in 1923 was of the opinion that intrauterine periarthritis might be looked upon as the etiologic agent in this syndrome. Lewin[3] in 1925 wrongly conceived that the joints were primarily at fault. Sheldon[4] in 1932 first put forward the idea that the muscles were at fault and confirmed his observations by investigation of the electrical reaction of the affected muscles, which showed no reaction of degeneration or weak or absent faradic responses. This proved that the muscle atrophy is not of peripheral nerve origin but indicates hypoplasia or complete absence of the muscles. He accordingly preferred the name "amyoplasia congenita," believing that the initial lesion is primarily aplasia or hypoplasia of certain muscle groups, the defects of the joints being secondary. Middleton[5] in 1932 used the term "myodystrophia fetalis deformans," stating that the deformities result from fatty degeneration of muscles during intrauterine life. Middleton found the muscles degenerated into fatty tissue and fibrous strands which contained only scant remnants of muscle fibers. There were irregular areas of the normal muscle fibers interwoven with fat and connective tissue. Some fields showed complete absence of muscle fibers with complete fibrous and fatty transformation. He expressed the belief that the muscular degeneration was analogous to the muscular dystrophies of later adult life in that the microscopic appearances are similar. The muscle degeneration of arthrogryposis arises fairly late in intrauterine life after the muscle fibers have been fully differentiated, and the condition progresses rapidly during late fetal life, ceasing at birth. The limb muscles are usually solely affected owing to the fact that the limb buds develop later than the trunk and arise from a different source.[5] Cook[6] in 1936 listed the following criteria for diagnosis:

1. Congenital joint rigidities of varying degrees, usually symmetric and always without inflammatory changes
2. Hypoplasia or degeneration of those muscles which usually produce the movements which the child or infant is unable to perform fully
3. Absence of any hypertonia and hypertrophy of antagonistic muscles, showing that immobility is not attributable to muscle overaction
4. Weak or absent faradic responses in affected muscles with no reaction of degeneration
5. No sensory or trophic changes
6. Normal intelligence
7. Extensive fibrofatty changes in the affected muscles.

Most published accounts of cases of arthrogryposis have reported clubfoot and clubhand in addition, but these are not necessary components. The outstanding feature is rigidity of one or more joints, but it never gives the impression of an absolute block. Unless there is an associated dislocation, the contracture is always springy no matter how greatly the motion may be restricted. The affected joints feel thickened and present a dimpling of the overlying skin creases. Proximal to the affected joint the extremity may have a sausagelike appearance due to hypertrophy of the subcutaneous fat, with little palpable muscle in the affected group. There seems to be a perfect relationship between the severity of the contractures and the degree of muscular degenerative changes. The limb may be contracted in extension or flexion, depending on the muscle group involved. There may be mixed flexion and extension contractures in different joints in the same individual.

Roentgenologic examinations made in the first few months of life show deficient muscle shadows with increased density in the capsular areas of the affected joints. In the x-ray picture taken early in life no gross osseous abnormalities will be apparent unless there is associated subluxation or dislocation. However, even in the absence of subluxation or dislocation, with long-standing contractures the affected articulations will tend to mushroom and spur, with eventual ankylosis.

A review of the literature failed to disclose any reports of dislocation of the knee attributed to arthrogryposis per se. However, McFarland[7] in 1929, reporting on a series of congenital dislocations of the knees, presented several cases which in addition to the knee disorders were associated with abnormal contractures of the elbows, feet, and/or hands. That these are unrecorded cases of arthrogryposis seems highly probable, as at that time little was known, or had been published, about this entity. Of these cases with associated joint rigidities conservative closed therapy directed to the dislocated knee was of little or no benefit and was soon abandoned in favor of operative procedures. McFarland's operative procedures consisted of open reductions, tenotomies, and capsulotomies of the knee followed with extensive physiotherapy. He records his results as fair to good if correction was done early, prior to secondary articular changes; if corrective procedures were delayed the outcome was inevitably poor.

CASE HISTORY AND EXAMINATION

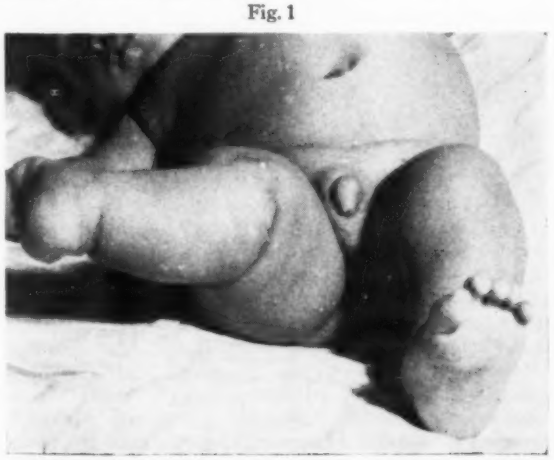
The maternal history showed three previous deliveries at this hospital, all uneventful births, and none of the babies had any congenital stigmata. This delivery on June 17, 1952, had not been an arduous one, the infant had cried immediately, and there had been no cyanosis or other evidence of fetal embarrassment. Feedings were started 12 hours following delivery and were well taken and tolerated. Note was made of the deformed right knee at the time of delivery, and an orthopedic consultation was requested on June 19, 1952.

Examination revealed a well-nourished, alert newborn male. The right lower extremity was rigidly held in an attitude of hyperextension and lateral deviation at the knee joint. The circumference of the right thigh was slightly larger than the left, and a mushy feeling over the anterior thigh musculature was noted. The skin did not appear abnormal, but the dimpling over the right knee was more pronounced than in other areas. The medial condyle of the right femur was quite prominent medially, with the tibia displaced anteriorly. No flexion of the right knee was possible, but a few additional degrees of "give" were present on forced hyperextension. The left knee appeared normal on casual inspection, but complete flexion was limited by approximately 20 degrees, and the infant could spontaneously dislocate the articulation so that it assumed a position similar to that of the right knee. With passive flexion the left knee would automatically reduce. There was no evidence of inflammation in either the joint or surrounding soft tissue, and light manipulation caused little discomfort. The remainder of the examination, including testing of the hip and of the mobility of the remaining joints, was noncontributory.

All laboratory findings were well within normal limits. Results of serologic tests were negative. Myelographic testing revealed weak faradic responses in both anterior thigh groups, more pronounced on the right and with no reaction of degeneration. X-rays indicated an anterolateral dislocation of the right tibia on the femur. The left knee, both hips, and remaining major joints showed no abnormalities according to the report of the roentgenologist.

Realizing that the dislocation should be reduced, and with the early reports of McFarland as a guide, we contemplated early surgery, but the mother of the infant would not consent to an operation, and we were forced to attempt a conservative approach. Continuous overhead traction, manipulations, corrective casting, and wedging were all unsuccessful in reducing the dislocated right knee. During this period the left knee was incorporated in the castings and progressively wedged. Following approximately 9 months of conservative therapy, dysplasia of the right acetabulum was discovered which subsequently resulted in dislocation of the hip. The mother was again approached regarding surgical intervention to the right knee and at this time consented to an operative procedure. Figure 1 shows the infant at this time; the right knee presented the same appearance as at birth.

Fig. 1



SURGERY

The operation was performed on June 24, 1953. With a right cephalic vein cutdown and with the infant under inhalation ether anesthesia, a medial parapatellar incision was made over the right knee. Blunt dissection revealed an atrophied and fibrotic quadriceps tendon and distal muscle belly, lying within the femoral condyles but displaced slightly laterally. The iliotibial band was taut and under excessive tension. Exposure of the joint revealed the tibia to be displaced anterolaterally on the femur. All anticular surfaces appeared normal, but the capsule was thickened. The cruciate ligaments were elongated and slender but intact. Following partial section of the taut iliotibial band near its insertion into the tibia, it was possible to reduce the knee so that opposing articular surfaces were congruent. Flexion remained limited, and following a Z-tenoplasty to lengthen the quadriceps tendon and a superior capsuloplasty of the anterior compartment, the knee could be flexed to approximately 90 degrees. There remained a moderate amount of lateral instability which was corrected by mobilizing and buttressing the sartorius tendon to the medial side of the proximal quadriceps tendon. Stability was further insured by anterior transplantation of the intact gracilis tendon to the periosteum and capsule on the anteromedial aspect of the distal femur. The knee no longer presented any instability at this stage and retained its articular relationships on flexion and extension. A Kirschner wire was then driven in a retrograde manner through the proximal tibia, traversing the knee joint, and into the distal femur to insure added stability during the healing phase. A bilateral spica cast was applied immediately following surgery. The convalescent period was uneventful. Skin sutures were removed in 2 weeks and the transfixion wire at the end of 6 weeks; the spica cast was left on for a total of 8 weeks. The spica cast was progressively wedged during the last 2 weeks, thus increasing the amount of flexion in both knees.

Examination following removal of the cast revealed the attitude and contour of the right knee to be quite satisfactory. Flexion was possible to 90 degrees, and the knee was stable in the medial and lateral planes. Extension was possible to 180 degrees, and there was no tendency to dislocate. Figure 2 shows the knee after the operation.

Attention was then turned to the dislocated right hip. Following a short period in which an abduction bar was used to stretch the contracted adductor mus-

Fig. 2

culature, closed reduction of the right hip was possible. A one and one-half spica was applied following reduction. At present the right hip is immobilized in plaster, and progressive wedging of the right knee joint is being performed in order to keep capsular shrinkage to a minimum.

CONCLUSION

The condition of the knee in this case resulted from a symmetric muscular degeneration of both anterior thigh groups. The advanced state of the pathologic condition in the right knee resulted from a more severe muscular degeneration—fibrosis, perhaps aided by the intrauterine position.

The vast majority of uncomplicated cases of arthrogryposis if recognized early are amenable to conservative therapy. The main obstacle in these cases is not degenerative atrophy of the musculature, but the secondary contractures of the musculature and joint capsule. The restoration of active motion naturally depends upon the state of the muscles at the time treatment is undertaken. With the static functions of standing and walking whatever muscle power is left will continue to develop so that the ability to bear weight will be present even with a minimum of muscle power. It is the extremely rare case in which complete muscle power is lost and, in this situation, musculotendinous transference is indicated. In the case described, the condition of the left knee is the intermediate stage between the uncomplicated and complicated types of arthrogryposis. This articulation has progressed favorably under conservative therapy consisting of casting and progressive wedging. The limitation of flexion which was present at birth is no longer present. The tendency to spontaneous dislocation on hyperextension no longer is present, but passive hyperextension will result in subluxation. Perhaps with additional conservative therapy the soft tissues will eventually accommodate to the point where any tendency to subluxation will be abolished.

The complicated case of arthrogryposis, associated with dislocation and/or secondary osseous changes of the articular surfaces, will inevitably require operative treatment to produce a functioning extremity. The types of surgical procedures will vary according to the degree and duration of the pathologic features. These procedures will include open reduction, tenoplasties, capsuloplasties, and, perhaps, osteotomy and arthroplasty in the more severe cases. In this case, the condition of the right knee was unchanged following 9 months of conservative therapy. At present, following operation, the outlook for a functioning extremity is much more favorable. The articulation now has approximately a 90-degree range of flexion and is stable; faradic testing reveals residual functioning musculature.

The question could then be asked, "What of the dislocation of the right hip in this patient when he was undergoing conservative treatment?" The fact that it was not dislocated at birth and there was no lack of joint mobility in the hip itself would rule out arthrogryposis as the etiologic agent. A review of the earlier x-rays revealed the right acetabular index to be slightly greater than the left, and thus the right hip is looked upon as a true congenital dysplasia-dislocation, having no direct relationship to the arthrogryposis. The early therapy (Bryant traction) and contracted anterior thigh musculature in all likelihood were important factors in the resultant dislocation of the right hip, but this was unavoidable since we had no choice but to attempt early conservative treatment of the right knee. If early surgery had been performed on the right knee, the possibility of development of a frank dislocation would have been greatly reduced.

SUMMARY

1. A case of bilateral knee rigidity, associated with a dislocation of one knee and a tendency of the other to dislocate, has been presented.

2. The possibility of arthrogryposis as the etiologic agent has been considered and a brief resume of this entity presented.

3. The course of treatment, complications, early results, and conclusions have been described.

1100 N. Mission Rd.

Acknowledgement

Sincere thanks are expressed to the Attending Staff of the Orthopedic Department of Los Angeles County General Osteopathic Hospital for their unselfish responses to my calls for help with orthopedic problems.

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DISCUSSION

A. E. Miller, Los Angeles: The call for help in this case did not produce any signs of genius in the beginning. I was the first on the attending staff of the Los Angeles County Osteopathic Hospital to see this patient. Without benefit of x-rays but with adequate clinical examination of this child, I made a diagnosis of congenital dislocation of the right knee and subluxation of the left. It remained for the senior on the service, Dr. Walter Garard, to diagnose the case correctly a few days later.

On examination of this child, there seemed to be a general laxity of the joint structure, which is certainly in direct contrast to the usual textbook description of arthrogryposis multiplex congenita. As a matter of fact, I argued the pros and cons of this case with myself for several weeks following, and it was some time before I became convinced that it was a case of arthrogryposis multiplex congenita. After seeing the patient and studying the literature, it seemed to me that this case more closely fitted the description of arthrochalarosis multiplex congenita that Hass[1] has described, the name of which he derived from the Greek words *arthron*, meaning joint, and *chalarosis*, meaning looseness. However, that this case has turned

out to be one of arthrogryposis multiplex congenita is now without question.

This paper has been well prepared and there is little that I can add as a commentator. However, there is one point that might profitably be discussed. Dr. Segal notes that according to the roentgenologist's report there was no abnormality of the hips. All too frequently we are too prone to accept the reports when as orthopedic surgeons we should be better qualified to interpret the films than the roentgenologist. The roentgenologist works at an extreme disadvantage; not only does he have to work under a tight schedule but also he is looking only at the x-ray films. He has no chance to examine the patient as well as the film as does the orthopedic surgeon. In a stack of films that the roentgenologist will view in one day, there are so many normal and so few abnormal findings that it is a wonder to me that he ever notices minimal abnormalities; certainly, a variation of a few degrees of the angle of a hip or any minimal variance in osseous structure would undoubtedly be overlooked. On the other hand, the orthopedist not only has the opportunity to view the films but also has the opportunity to correlate these films with the clinical findings. In such a case as this, there should be more than a suspicion of the possibilities of other congenital problems and in viewing the films the orthopedist should be actually looking for pathologic findings.

In reviewing the original x-rays of the pelvis, these few degrees of variation can be seen. That this was overlooked by the roentgenologist does not surprise me, inasmuch as the orthopedist also overlooked it. In reading reports of negative findings, we may possibly complacently sit by and allow dislocations of hips to occur later in life that need not necessarily have occurred if therapy had been instituted early. True, we might overcompensate and wrongly interpret some things as pathologic; however, this would be infrequent. This overcompensation might be very good, causing us at least to follow cases more closely and view our clinical examination more critically in an attempt to prove the suspicion we have in our minds after viewing films.

I would disagree with Dr. Segel in his statement that arthrogryposis is not the agent etiologic in the dislocation of the right hip. I do not believe this was typical congenital dysplasia of the hip that became apparent on dislocation due to traction, casts, et cetera, used in the treatment of the knees. I would ask Dr. Segel two things: (1) Was closed reduction obtained easily as in typical hip dysplasia? (2) If this reduction was obtained, was it as easily held in the simple abduction plaster cast? According to what I heard about the progress of this case, the hip not only was difficult to reduce but also was difficult to maintain in a normal attitude. In my opinion, dislocation of the hip was merely another feature of arthrogryposis.

Having seen this patient in the beginning and misdiagnosed his condition, and having heard of the progress of the case, at least in part, it has been a pleasure to have been permitted to take part in this discussion. The case has been most instructive.

766 S. Kingsley Drive

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Review of H Graft Lumbar Fusions

CHESTER H. LYON, D.O.
Los Angeles

The most common single ailment confronting the orthopedic surgeon today is the low-back problem with its multiplicity of variations in diagnosis and treatment. These complaints extend from mild occasional strain with associated muscle spasm to the acute problem with sciatic scoliosis and extensive sciatic neuritis. It is the patient with this latter type of involvement who so often ends up in the hands of the neurologist and is operated upon for removal of a disk. Unfortunately many of these patients are relieved for a short time only and then the same symptomatology recurs, so that dissatisfaction results, and there is a tendency toward doubt when relief is again offered.

In my practice I have utilized knowledge of these problems in the following manner: If the case is of a mild nature I institute physiotherapy, osteopathic manipulative therapy, and home exercises. Of course, any postural derangements, obesity, short leg, and so on are corrected. Usually these measures suffice to give relief and stability to the patient's lower back.

If the condition is semiacute and if there is a history of previous episodes at long intervals, I find that a regimen which includes traction in bed, either by alternating Russell traction for 2 hours with 2 hours without traction during the waking hours, or the use of the Camp-Varco pelvic belt with continuous traction during the waking hours, reduces the associated muscle spasm and allows manipulation with ease. Following this procedure, the wearing of a Williams back brace is continued for a time. Occasionally I find that the muscle spasm has not been relieved by traction and will respond only to nonsurgical reduction or osteopathic manipulative therapy with the patient under anesthesia, in which case a Williams type back brace is also employed postoperatively.

Next, there is the patient who presents himself at frequent intervals with an acute problem which responds only to long-drawn-out office treatments or repeated traction and osteopathic manipulative therapy. He even has recurrences while wearing some support. This type of case is best aided by surgical intervention, and the patient is so advised.

Finally, there is the acute problem with associated sciatic neuritis and intractable pain which usually has recurred at frequent intervals. Such intervals are closer and closer together as time advances. This is the patient who demands immediate relief and, although traction relieves the spasm, the neuritic pain persists for a long time after the anatomic derangement of the spine has been corrected. This is another type of case for which I advise surgery as the treatment of choice.

At this time, I shall review my choice of surgery, the procedure, and aftercare for these cases and evaluate statistically a number of these cases treated in the past few years.

All orthopedists know the role of trauma in the production of these low-back problems. Unfortunately, the present-day regimen of sports for the adolescent, failure to instruct workers in the proper methods of lifting and carrying, failure to teach mothers how to handle children as they grow up, and accidents associated with high-speed civilization, all produce one condition; that condition is tearing or injury to the ligamentous structures which maintain the integrity and continuity of the spinal column. Since the lower back, the lumbosacral spine, is the apex of such forces, it is only to be expected that the greatest number of such incidents and injuries will occur in this area. It is my opinion that this ligamentous damage, more than structural or embryonic anomalies, constitutes the etiologic agent in the formation of low-back problems.

In surgical repair of this injury, I use the H type of fusion which, in effect, acts as a strut and thus bypasses the function of the ligaments and at the same time increases the stability and spreads the apical force over two or more components of the vertebral column.

I have evolved a transverse incision of the reverse type over the lumbosacral junction from the posterior iliac spine to the opposite posterior iliac spine. The patient is placed prone on the table with either the Wilson type of support or sandbags at each anterior superior iliac spine and at the thorax to prevent compression of the abdomen, the break in the table being at the level of the planned surgery. The choice of anesthesia is usually Pentothal sodium induction with intubation and the use of ether, gas, and Pentothal sodium or gas exclusively. Transfusions are always provided for during surgery and postsurgically to minimize the shock syndrome associated with this procedure.

After exposure of the spinous processes through the transverse skin incision, hemostasis is secured in the skin and subcutaneous tissues by coagulation with the Birtcher coagulator. The muscles are stripped away on either side of the spinous processes with a large osteotome, and these spaces are packed with hot, moist lap sponges. Then I denude the spinous processes by sharp dissection, removing the posterior intervertebral ligaments and the soft tissues between the spinous processes. I next identify the vertebral levels and cleanse the spinous processes and lamina of all soft tissue remainders. Usually at this point I test the mobility of the exposed vertebrae with Lion bone forceps, and the mobility of the affected vertebral component or components is surprising. The freeing of the spinous processes allows greater mobility, but even without this freeing there is still greater mobility in the affected vertebrae than in the intact vertebrae above or below. Whether or not I perform hemilaminectomy at this point will depend on the amount of nerve root pressure present on examination.

The spinous processes of the one or two vertebrae which are to be fused are then split and laid down over the lamina on either side. The vertebrae above and below are notched and the whole spinous area is packed with hot sponges to promote hemostasis. The iliac crest just lateral to the sacroiliac joint is easily reached through the original incision.

A suitable double-thickness area of the crest is denuded of soft tissue and removed with the chisel and mallet. The assistant removes cancellous bone from the site of the graft. The graft is then measured, notched at its ends, and the patient's body is acutely flexed on the table by dropping the head and feet; the graft is placed in position in the notched spinous processes. The patient's body is then returned to the straight prone position which should automatically lock the graft into position so that it cannot be moved by the operator's fingers. Loose cancellous bone is placed around the graft close to the raw surface which is in contact with the raw surfaces of the spinous processes.

Routine closure of the incision is made over the site of the graft and over the spine with double 0 chromic catgut. The skin is closed with interrupted black silk sutures, and dry sterile dressings are applied and held in position with elastoplast dressing.

The patient is carefully returned to bed and placed in the supine position and remains in that position for 3 to 5 days. A blanket roll or pillow is allowed under the knees. Transfusion is ordered immediately after the patient is returned to bed; sedatives, antibiotics, and the usual hospital routine care are prescribed. The wound is dressed on the fifth postoperative day, a dorsolumbar corset is provided, and the patient is ambulated. The patient usually leaves the hospital between the eighth and tenth postoperative day.

The follow-up regimen consists of continuous wearing of the corset, day and night, for 1 month, changing of dressings, and monthly x-rays for evaluation of the bone changes. The dorsolumbar support is worn only during waking hours after the first month. It is discarded on discharge. The patient is usually discharged 6 months after surgery. Follow-up care is provided at 6-month intervals for evaluation of results.

A statistical survey of 80 cases followed for a period of 1 to 3 years revealed the following: Males comprised 85 per cent of the cases, females 15 per cent. Occupations included plumber, longshoreman, clerk, truck driver, bookkeeper, executive, and housewife. Of this group, all were able to return to the full range of duties of their previous occupations. Two patients reported poor results or continued disability and former symptoms. Eight reported some sciatic pain on the previously affected side after heavy exertion or long ambulation, but they stated they felt the results were satisfactory. Seventy patients reported excellent results with no disability, but one of this group reported tenderness on pressure over the scar area. One had persistent postoperative drainage for 8 weeks until a culture was taken and sensitivity of the organisms to antibiotics was demonstrated. After heavy dosage of the antibiotic was given drainage ceased. Drainage later recurred, excision of the sinus was performed, and healing was uneventful, although the scar was not satisfactory from a cosmetic standpoint.

The length of time for return to work ranged from 2 weeks postoperatively in a female patient whose occupation was rug sewing to 16 weeks in the heavy labor classes, such as shipping clerk and longshoreman. The average time between surgery and return to full employment was 6.8 weeks.

SUMMARY

An evaluation of one surgical method for treatment of persistent low-back pain has been given. The results of the statistical survey and follow-up—the relatively short hospitalization, the short period of disability, and the high percentage of satisfactory results—have been encouraging.

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Tuesday, July 5

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
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8:00- 9:00 Registration and Indoctrination
9:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons
1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Peripheral Venous Problems — Etiology, Symptomatology, and Diagnosis
John A. Costello, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 6

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons
1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Peripheral Venous Problems—Treatment
William T. Barrows, D.O., Oakland, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Thursday, July 7

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons
1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Peripheral Arterial Problems — Etiology, Symptomatology, and Diagnosis
Joseph O. Costello, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Friday, July 8

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles
2:00 Peripheral Arterial Problems—Treatment
Donald T. Sheldon, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Monday, July 11

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles
2:00 Great Vessel Problems—Diagnosis and Treatment
W. Donald Baker, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Tuesday, July 12

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Congenital Cardiovascular Problems—Symptomatology and Diagnosis
William D. Long, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 13

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Congenital Cardiovascular Problems—Treatment (Medical and Surgical)
William D. Long, D.O., Los Angeles
J. Willoughby Howe, D.O., Hollywood, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Thursday, July 14

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Acquired Cardiovascular Problems—Symptomatology and Diagnosis
Glennard E. Lahrson, D.O., Oakland, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Friday, July 15

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pathology
Ray L. McClure, D.O., Los Angeles
2:00 Acquired Cardiovascular Problems—Treatment (Medical and Surgical)
Glennard E. Lahrson, D.O., Oakland, Calif.
W. Donald Baker, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Saturday, July 16

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pathology
Ray L. McClure, D.O., Los Angeles
2:00 Essential Hypertension—Diagnosis and Treatment (Surgical)
Robert K. Schieffer, D.O., San Diego, Calif.
W. Donald Baker, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

SECTION II—REGULAR SURGERY

This course is to be limited to members or candidates of the American College of Osteopathic Surgeons or individuals eligible to the College. Registrants will be required to scrub and act as surgical assistants for numerous types of vascular surgical procedures to be performed each morning in various hospitals in the Los Angeles area. In addition, the registrants will act as assistants to the surgeons of Section I, thereby completing four-man surgical teams for proper training in upper abdominal, thoracoabdominal, cardiovascular, and peripheral vascular surgical technics on the experimental animal. These registrants will attend the same lecture session as those registered in Section I.

Section limited to 20 students.....Fee $150.00

FACULTY FOR SECTION II—REGULAR SURGERY

J. Gordon Hatfield, D.O., Los Angeles
HEAD OF THE DEPARTMENT OF SURGERY, COLLEGE OF OSTEOPATHIC PHYSICIANS AND SURGEONS
W. Donald Baker, D.O., Los Angeles
DIRECTOR OF SURGICAL COURSES
John A. Costello, D.O., Los Angeles
COORDINATOR OF HOSPITAL SURGERY
William T. Barrows, D.O., Oakland, Calif.
COORDINATOR OF ANIMAL SURGERY

CLINICAL SURGICAL FACULTY

W. Donald Baker, D.O., Los Angeles
William T. Barrows, D.O., Oakland, Calif.
Wilbur L. Brandt, M.A., Los Angeles
John A. Costello, D.O., Los Angeles
Joseph O. Costello, D.O., Los Angeles
David W. Dale, D.O., Seattle
Robert W. Earle, M.S., Los Angeles
A. Kent Darrow, D.O., North Hollywood, Calif.
J. Gordon Hatfield, D.O., Los Angeles
LaMoyne W. Hickman, D.O., Los Angeles
J. Willoughby Howe, D.O., Hollywood, Calif.
A. Verne Jackson, D.O., Forest Grove, Ore.
Delbert F. Johnson, D.O., Seattle
Walter E. Mattocks, D.O., Long Beach, Calif.
Ray L. McClure, D.O., Los Angeles
William E. Merrill, D.O., Seattle
Ira J. Neher, D.O., Portland, Ore.
Donald T. Sheldon, D.O., Los Angeles
William G. Stahl, D.O., Pomona, Calif.
Richard R. Stuart, Ph.D., Los Angeles

Tuesday, July 5

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
Hospitals in Los Angeles Area

8:00- 9:00 Registration and Indoctrination
9:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Peripheral Venous Problems — Etiology, Symptomatology, and Diagnosis
John A. Costello, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 6

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Peripheral Venous Problems—Treatment
William T. Barrows, D.O., Oakland, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Thursday, July 7

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Peripheral Arterial Problems — Etiology, Symptomatology, and Diagnosis
Joseph O. Costello, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Friday, July 8

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles
2:00 Peripheral Arterial Problems—Treatment
Donald T. Sheldon, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Monday, July 11

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles
2:00 Great Vessel Problems—Diagnosis and Treatment
W. Donald Baker, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Tuesday, July 12

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Congenital Cardiovascular Problems—Symptomatology and Diagnosis
William D. Long, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 13

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Congenital Cardiovascular Problems—Treatment (Medical and Surgical)
William D. Long, D.O., Los Angeles
J. Willoughby Howe, D.O., Hollywood, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Thursday, July 14

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Acquired Cardiovascular Problems—Symptomatology and Diagnosis
Glennard E. Lahrson, D.O., Oakland, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Friday, July 15

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pathology
Ray L. McClure, D.O., Los Angeles
2:00 Acquired Cardiovascular Problems—Treatment (Medical and Surgical)
Glennard E. Lahrson, D.O., Oakland, Calif.
W. Donald Baker, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Saturday, July 16

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pathology
Ray L. McClure, D.O., Los Angeles
2:00 Essential Hypertension—Diagnosis and Treatment (Surgical)
Robert K. Schieffer, D.O., San Diego, Calif.
W. Donald Baker, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

SECTION III—INTERNAL MEDICINE AND CARDIOLOGY

This course is to be limited to osteopathic physicians and surgeons who are members or candidates of the American College of Osteopathic Internists or are eligible to the College. Each morning complete and extensive cardiovascular diagnostic clinics will be held at the Los Angeles County Osteopathic Hospital which will enable registrants to examine and take part actively in the general medical work-up of each case. All of the resources of the Los Angeles County Osteopathic Hospital Department of Internal Medicine and Cardiology will be made available, together with many of the attending staff as clinical instructors. In addition there will be afternoon sessions dealing with basic sciences, internal medicine, and cardiology.

Cardiac catheterization and angiocardiography will also be featured as part of the training.

Section limited to 40 students.....Fee $150.00

FACULTY FOR SECTION III—INTERNAL MEDICINE AND CARDIOLOGY

Lawrence B. O'Meara, D.O., Los Angeles
HEAD OF THE DEPARTMENT OF INTERNAL MEDICINE AND CARDIOLOGY, COLLEGE OF OSTEOPATHIC PHYSICIANS AND SURGEONS

Munish Feinberg, D.O., Los Angeles
DIRECTOR OF INTERNAL MEDICINE AND CARDIOLOGY COURSE

J. Claver Cargill, D.O., Burbank, Calif.
COORDINATOR OF COLLEGE CARDIOVASCULAR DIAGNOSTIC CLINICS

CLINICAL FACULTY

S. J. Aquilla, D.O., Van Nuys, Calif.
Wilbur L. Brandt, M.A., Los Angeles
Maxwell R. Brothers, D.O., Los Angeles
J. Claver Cargill, D.O., Burbank, Calif.
Stanley H. Cowell, D.O., Los Angeles
Delmar J. Daniel, D.O., Burbank, Calif.
Robert W. Earle, M.S., Los Angeles
W. H. Enenstein, D.O., Los Angeles
Munish Feinberg, D.O., Los Angeles
Chester F. Foster, D.O., Los Angeles
Milton S. Futterman, D.O., Los Angeles
Orville L. Hastings, D.O., Long Beach, Calif.
Aaron H. Jaffe, D.O., Los Angeles
Victor Kovner, D.O., North Hollywood, Calif.
Glennard E. Lahrson, D.O., Oakland, Calif.
William D. Long, D.O., Los Angeles
Ray L. McClure, D.O., Los Angeles
Paul B. McCracken, D.O., Pasadena, Calif.
R. F. Nickson, D.O., Los Angeles
C. L. Nye, D.O., Los Angeles
Lawrence B. O'Meara, D.O., Los Angeles
Donald E. Pinder, D.O., Los Angeles
Homer E. Randolph, D.O., Los Angeles
J. Holt Robison, D.O., Los Angeles
Richard A. Schaub, D.O., Pasadena, Calif.
Robert K. Schieffer, D.O., San Diego, Calif.
Hermon H. Schlossberg, D.O., Los Angeles
Ernest L. Stebbins, D.O., Sherman Oaks, Calif.
Harold J. Strick, D.O., Los Angeles
Richard R. Stuart, Ph.D., Los Angeles
John A. Tedford, D.O., Burbank, Calif.
Ernest B. Van Horn, D.O., South Pasadena, Calif.

Tuesday, July 5

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
Hospitals in Los Angeles Area

8:00- 9:00 Registration and Indoctrination
9:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Electrolyte Balance
Victor Kovner, D.O., North Hollywood, Calif.
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 6

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Electrocardiography
Harold J. Strick, D.O., Los Angeles
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Thursday, July 7

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 The Treatment of Cardiac Emergencies
Munish Feinberg, D.O., Los Angeles
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Friday, July 8

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles
2:00 The Treatment of Thyrotoxic Heart Disease
Maxwell R. Brothers, D.O., Los Angeles
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Monday, July 11

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles
2:00 X-Ray Diagnosis of Heart Disease
J. Holt Robison, D.O., Los Angeles
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Tuesday, July 12

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles

2:00 Congenital Cardiovascular Problems—Symptomatology and Diagnosis
William D. Long, D.O., Los Angeles
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 13

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons
1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Congenital Cardiovascular Problems—Treatment (Medical and Surgical)
William D. Long, D.O., Los Angeles
J. Willoughby Howe, D.O., Hollywood, Calif.
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Thursday, July 14

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons
1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Acquired Cardiovascular Problems—Symptomatology and Diagnosis
Glennard E. Lahrson, D.O., Oakland, Calif.
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Friday, July 15

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons
1:00 Pathology
Ray L. McClure, D.O., Los Angeles
2:00 Acquired Cardiovascular Problems—Treatment (Medical and Surgical)
Glennard E. Lahrson, D.O., Oakland, Calif.
W. Donald Baker, D.O., Los Angeles
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Saturday, July 16

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Cardiovascular Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons
1:00 Pathology
Ray L. McClure, D.O., Los Angeles
2:00 Essential Hypertension — Diagnosis and Treatment (Surgical)
Robert K. Schieffer, D.O., San Diego, Calif.
W. Donald Baker, D.O., Los Angeles
3:00- 5:00 Diagnostic and Therapeutic Clinics
5:00- 7:00 Dinner Period

SECTION IV—GENERAL PRACTICE AND PERIPHERAL VASCULAR DISEASE

This course is designed for all general practitioners in osteopathic medicine and surgery. Any member of the American Osteopathic Association is eligible. The registrants of this section will be divided into three groups which will rotate daily in the mornings:

1. Surgical observation of cardiovascular procedures.
2. Observation and taking part in cardiovascular clinics.
3. Observation and taking part in peripheral vascular clinics at the Los Angeles County Osteopathic Hospital.

Each afternoon will be devoted to basic science and didactic lectures designed to give the registrant a thorough grounding in many phases of general practice, including cardiovascular and peripheral vascular diseases. In addition, the different section will attend sessions at the College of Osteopathic Physicians and Surgeons Rehabilitation Center, at which clinical cases will be demonstrated. Included in these demonstrations will be patients with such diseases as poliomyelitis, muscular dystrophy, arthritis, and hemiplegia following cerebrovascular accidents. The Speech Clinic will be available to registrants, and technics for the care and training of patients with speech problems will be demonstrated. Another phase of instruction will cover various osteopathic manipulative technics and the use of such modalities as mechanical and traction tables, ultrasonic therapy, and electromyography in the diagnosis and treatment of muscular disease. Each afternoon the different sections will be rotated as follows:

1. Demonstration of clinical cases at rehabilitation center and manipulative section at the College of Osteopathic Physicians and Surgeons.
2. Observation of cardiovascular technical procedures such as angiocardiography and cardiac catheterization.
3. Therapeutic clinics in office procedures related to the care of general practice cases and peripheral vascular cases at the College of Osteopathic Physicians and Surgeons General Clinic.

Section limited to 150 students.....Fee $75.00

FACULTY FOR SECTION IV—GENERAL PRACTICE AND PERIPHERAL VASCULAR DISEASE

John V. Fiore, D.O., Los Angeles
DIRECTOR OF GENERAL PRACTICE COURSE
Walter E. Mattocks, D.O., Long Beach, Calif.
COORDINATOR OF LOS ANGELES COUNTY OSTEOPATHIC HOSPITAL DIAGNOSTIC PERIPHERAL VASCULAR CLINICS
Donald T. Sheldon, D.O., Los Angeles
COORDINATOR OF COLLEGE OF OSTEOPATHIC PHYSICIANS AND SURGEONS THERAPEUTIC PERIPHERAL VASCULAR CLINICS
John M. Andrews, D.O., Los Angeles
COORDINATOR OF THE COLLEGE OF OSTEOPATHIC PHYSICIANS AND SURGEONS REHABILITATION AND MANIPULATION THERAPY CLINICS

CLINICAL FACULTY

John M. Andrews, D.O., Los Angeles
W. Donald Baker, D.O., Los Angeles
William T. Barrows, D.O., Oakland, Calif.
Martin C. Beilke, D.O., Chicago
Wilbur L. Brandt, M.A., Los Angeles
John A. Costello, D.O., Los Angeles
Joseph O. Costello, D.O., Los Angeles
J. S. Denslow, D.O., Kirksville, Mo.
Irving Jay Dunn, D.O., Los Angeles
Robert W. Earle, M.S., Los Angeles
Bertha Evans, D.O., Los Angeles
John V. Fiore, D.O., Los Angeles
Norman Freestone, Ph.D., Los Angeles
LaMoyne W. Hickman, D.O., Los Angeles
David Heilig, D.O., Philadelphia
J. Willoughby Howe, D.O., Los Angeles
Wallin W. King, D.O., Los Angeles
Walter E. Mattocks, D.O., Long Beach, Calif.
Ray L. McClure, D.O., Los Angeles
Donald E. Pinder, D.O., Los Angeles
William W. W. Pritchard, D.O., Los Angeles
John A. Schuck, D.O., Los Angeles
Donald T. Sheldon, D.O., Los Angeles
D. Duane Stonier, D.O., Los Angeles
Richard R. Stuart, Ph.D., Los Angeles

Tuesday, July 5

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons Hospitals in Los Angeles Area

8:00- 9:00 Registration and Indoctrination

9:00-12:00 Clinics

This class will be divided into three groups, which will rotate daily on an observational basis as follows:

1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)
3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital)

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles

2:00 Peripheral Venous Problems — Etiology, Symptomatology and Diagnosis
John A. Costello, D.O., Los Angeles

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:

1. Demonstration of clinical cases at Rehabilitation Center and Manipulative Section at the College of Osteopathic Physicians and Surgeons
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 6

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Clinics

This class will be divided into three groups, which will rotate daily on an observational basis as follows:

1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)
3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles

2:00 Peripheral Venous Problems—Treatment
William T. Barrows, D.O., Oakland, Calif.

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:

1. Demonstration of clinical cases at Rehabilitation Center and Manipulative Section at the College of Osteopathic Physicians and Surgeons
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Thursday, July 7

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Clinics

This class will be divided into three groups, which will rotate daily on an observational basis as follows:

1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)
3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles

2:00 Peripheral Arterial Problems — Etiology, Symptomatology, and Diagnosis
Joseph O. Costello, D.O., Los Angeles

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:
1. Demonstration of clinical cases at Rehabilitation Center and Manipulative Section at the College of Osteopathic Physicians and Surgeons
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Friday, July 8

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Clinics
This class will be divided into three groups, which will rotate daily on an observational basis as follows:
1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)
3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital)

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles

2:00 Peripheral Arterial Problems—Treatment
Donald T. Sheldon, D.O., Los Angeles

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:
1. Animal Surgery (College and Graduate School)
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Monday, July 11

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Clinics
This class will be divided into three groups, which will rotate daily on an observational basis as follows:
1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)
3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital)

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles

2:00 Great Vessel Problems — Diagnosis and Treatment
W. Donald Baker, D.O., Los Angeles

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:
1. Animal Surgery (College and Graduate School)
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Tuesday, July 12

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Clinics
This class will be divided into three groups, which will rotate daily on an observational basis as follows:
1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)
3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital)

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles

2:00 Psychosomatic Medicine
Irving Jay Dunn, D.O., Los Angeles

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:
1. Animal Surgery (College and Graduate School)
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 13

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Clinics
This class will be divided into three groups, which will rotate daily on an observational basis as follows:
1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)

3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital)

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles

2:00 Renal Hypertension
D. Duane Stonier, D.O., Los Angeles

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:
1. Animal Surgery (College and Graduate School)
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Thursday, July 14

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Clinics
This class will be divided into three groups, which will rotate daily on an observational basis as follows:
1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)
3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital)

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles

2:00 Muscular Physiology
J. S. Denslow, D.O., Kirksville, Mo.

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:
1. Animal Surgery (College and Graduate School)
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Friday, July 15

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Clinics
This class will be divided into three groups, which will rotate daily on an observational basis as follows:
1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)
3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital)

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pathology
Ray L. McClure, D.O., Los Angeles

2:00 Manipulative Therapy
Martin C. Beilke, D.O., Chicago
David Heilig, D.O., Drexel Hill, Pa.

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:
1. Animal Surgery (College and Graduate School)
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Saturday, July 16

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Clinics
This class will be divided into three groups, which will rotate daily on an observational basis as follows:
1. Surgical Clinics (Hospitals in Los Angeles Area)
2. Peripheral Vascular Clinics (Los Angeles County Osteopathic Hospital)
3. Cardiovascular Clinics (Los Angeles County Osteopathic Hospital)

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pathology
Ray L. McClure, D.O., Los Angeles

2:00 Practical Neurology
Donald E. Pinder, D.O., Los Angeles

3:00- 5:00 Three groups will rotate daily on an observational basis as follows:
1. Animal Surgery (College and Graduate School)
2. Peripheral Vascular Clinics — Office Procedures
3. Diagnostic and Therapeutic Clinics

5:00- 7:00 Dinner Period

SECTION V—ANESTHESIOLOGY

This course will be limited to members of the osteopathic profession who are either certified in anesthesiology, in training for such certification, or are spending a portion of their time giving anesthetics and are interested in improving their skill and learning new anesthetic technics.

Each morning will be spent in the various hospitals in the Los Angeles area working with the staff anesthetists in the actual administration of all types of anesthesia as material presents.

Each afternoon will be spent at suitable basic science and didactic lectures.

These lectures will be followed by animal surgical procedures in which the registrants will be the anesthetic procedures.

Section limited to 40 students.....Fee $150.00

FACULTY FOR SECTION V—ANESTHESIOLOGY

Glenn F. Gordon, D.O., Glendale, Calif.
HEAD OF ANESTHESIA DEPARTMENT, COLLEGE OF OSTEOPATHIC PHYSICIANS AND SURGEONS
Glenn F. Gordon, D.O., Glendale, Calif.
DIRECTOR OF ANESTHESIA COURSE
John B. Quick, D.O., Van Nuys, Calif.
COORDINATOR OF HOSPITAL ANESTHESIA
Lowell H. Theissen, D.O., Gardena, Calif.
COORDINATOR OF ANIMAL ANESTHESIA

CLINICAL ANESTHETIC FACULTY

Bernard Adland, D.O., South Gate, Calif.
John C. Bell, D.O., Los Angeles
Rosalyn L. Binning, D.O., Whittier, Calif.
Wilbur L. Brandt, M.A., Los Angeles
S. R. Daniels, D.O., Los Angeles
Robert W. Earle, M.S., Los Angeles
Carl Eyerick, D.O., Burbank, Calif.
Glenn F. Gordon, D.O., Glendale, Calif.
Robert L. Harmon, Jr., D.O., Los Angeles
Kenneth R. Holcomb, D.O., Hollywood, Calif.
Harold C. Holloway, D.O., Los Angeles
James W. Jensen, D.O., Long Beach, Calif.
Lowell W. Kearl, D.O., Los Angeles
Fred T. Lowrie, D.O., Gardena, Calif.
Dorothy J. Marsh, D.O., Los Angeles
Ray L. McClure, D.O., Los Angeles
Claire E. Pike, D.O., Long Beach, Calif.
John B. Quick, D.O., Van Nuys, Calif.
John S. Stratton, D.O., Alhambra, Calif.
Richard R. Stuart, Ph.D., Los Angeles
Afton C. Taylor, D.O., Norwalk, Calif.
Lowell H. Theissen, D.O., Gardena, Calif.
Seymour Ulansey, D.O., Los Angeles

Tuesday, July 5

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
Hospitals in Los Angeles Area

8:00- 9:00 Registration and Indoctrination
9:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Principles of Balanced Anesthesia
James W. Jensen, D.O., Long Beach, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 6

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Cardiac Arrhythmias under Anesthesia
John B. Quick, D.O., North Hollywood, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Thursday, July 7

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Anatomy
Richard R. Stuart, Ph.D., Los Angeles
2:00 Etiology of and Precautions Against Cardiac Arrest
Lowell W. Kearl, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Friday, July 8

MORNING SESSION

Hospitals in Los Angeles Area
8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles
2:00 Hazards of Anesthesia for Pelvic Delivery
Dorothy J. Marsh, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons
7:00- 9:00 Visual Education Program and Seminar

Monday, July 11

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Physiology
Wilbur L. Brandt, M.A., Los Angeles
2:00 Blood Transfusion—Indications and Contraindications—Arterial vs. Venous and Untoward Reaction in the Anesthetized Patient
Carl Eyerick, D.O., Burbank, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Tuesday, July 12

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Evaluation of the Various Technics Relative to Efficient Respiratory Exchange in Pediatric Anesthesia
Afton C. Taylor, D.O., Norwalk, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Wednesday, July 13

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Contraindications and Complications of Endotracheal Anesthesia
Lowell H. Theissen, D.O., Gardena, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Thursday, July 14

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pharmacology
Robert W. Earle, M.S., Los Angeles
2:00 Conduction Anesthesia in Traumatic Surgery of the Lower Extremities
Robert L. Harmon, Jr., D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Friday, July 15

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pathology
Ray L. McClure, D.O., Los Angeles
2:00 Discussion of Cortisone as Related to Anesthesia
Glenn F. Gordon, D.O., Glendale, Calif.
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Visual Education Program and Seminar

Saturday, July 16

MORNING SESSION

Hospitals in Los Angeles Area

8:00-12:00 Surgical Clinics
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Pathology
Ray L. McClure, D.O., Los Angeles
2:00 Cardiology in Anesthesiology
S. R. Daniels, D.O., Los Angeles
3:00- 5:00 Animal Surgery
5:00- 7:00 Dinner Period

SPECIAL COURSE— PSYCHOPHYSIOLOGIC MEDICINE

A practical course for the general practitioners who wish to attain greater effectiveness in the diagnosis and treatment of problem patients whose emotional maladjustment initiates or intensifies symptomatic manifestations. The new advances in neuroanatomy and neurophysiology and their relationship to psychophysiologic autonomic and visceral reactions will be presented and discussed. Diagnostic and therapeutic methods applicable to office practice will be emphasized. All didactic material will be integrated so the physician gains a comprehensive knowledge of the interrelated factors responsible for mental illness.

Then, following the above outlined course, a 2-day symposium on hypnosis will be conducted. This course is designed to give the practicing physician a general knowledge of the nature of hypnosis and the methods of induction, along with a survey of its application to medical problems. Actual demonstrations will be conducted and opportunity given each student to practice induction technic, under supervision.

The course will be presented in 2 full days. The first session will be a lecture including general orientation, history, and theory, followed by answering questions from the class membership. The remaining session will be in small groups with an instructor for each group. Here the actual technic of induction will be taught and practiced by the members of the class. In all the sessions, the members of the class will be expected to participate actively.

Indications and contraindications and the various technics possible under hypnosis, that is, diagnostic investigation, hypnoanalysis, anesthesia, sedation, symptom removal, et cetera, will be discussed and demonstrated. Practical teaching sessions in technic will be on the basis of one instructor to each ten members of the class.

This course is designed for all general practitioners in osteopathic medicine and surgery. Any member of the American Osteopathic Association is eligible.

Classes will be conducted at the College of Osteopathic Physicians and Surgeons, Los Angeles.

Checks should be made payable to: Graduate School, College of Osteopathic Physicians and Surgeons.

Section limited to 40 students.....Fee $75.00

FACULTY FOR SPECIAL COURSE— PSYCHOPHYSIOLOGIC MEDICINE

Don C. Littlefield, D.O., Long Beach, Calif.
COURSE DIRECTOR

CLINICAL FACULTY

Karl L. Albaeck, D.O., Burbank, Calif.
K. Grosvenor Bailey, D.O., Los Angeles
Floyd E. Dunn, D.O., Kansas City, Mo.
Leonard D. Gluckson, D.O., Los Angeles
Paul B. Harbour, D.O., Milton, Mass.
Oscar Janiger, D.O., Los Angeles
Don C. Littlefield, D.O., Long Beach, Calif.
Ralph I. McRae, D.O., Dallas, Tex.
Bernauer W. Newton, Ph.D., Los Angeles
David H. Payne, D.O., Los Angeles
Edwin F. Peters, Ph.D., Des Moines, Iowa
Donald E. Pinder, D.O., Los Angeles
Sidney D. Rothman, D.O., Los Angeles

Monday, July 11

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

8:00 Course Orientation
Don C. Littlefield, D.O., Long Beach, Calif.

9:00 Personality Development
1. Normal: Infancy-Adult-Puberty-Senile
2. Organic Deterrents to Normal Development: Brain Injury vs. Behavior Problems
Ralph I. McRae, D.O., Dallas, Tex.

10:00 Stress—Selye Theory
Oscar Janiger, D.O., Los Angeles

11:00 Anxiety

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Unconscious Motivation and Psychophysiologic Reaction
Paul B. Harbour, D.O., Milton, Mass.

2:00 Nervous Pathways—Anatomy
Donald E. Pinder, D.O., Los Angeles

3:00 Nervous Pathways—Neurophysiology
Karl L. Albaeck, D.O., Burbank, Calif.

3:30 Psychophysiologic Autonomic and Visceral Disorders:
001-580—Psychophysiologic Skin Reaction
002-580—Psychophysiologic Musculoskeletal Reaction
Kenneth C. Brandenburg, M.D., Long Beach, Calif.
Floyd E. Dunn, D.O., Kansas City, Mo.
Sidney D. Rothman, D.O., Los Angeles

5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Psychophysiologic Autonomic and Visceral Disorders:
003-580—Psychophysiologic Respiratory Reaction
004-580—Psychophysiologic Cardiovascular Reaction
005-580—Psychophysiologic Hemic and Lymphatic Reaction
Kenneth C. Brandenburg, M.D., Long Beach, Calif.
Floyd E. Dunn, D.O., Kansas City, Mo.
Sidney D. Rothman, D.O., Los Angeles

Tuesday, July 12

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

8:00 006-580—Psychophysiologic Gastrointestinal Reaction
007-580—Psychophysiologic Genitourinary Reaction
008-580—Psychophysiologic Endocrine Reaction
009-580—Psychophysiologic Nervous System Reaction
00x-580—Psychophysiologic Reaction of Organs of Special Sense
Kenneth C. Brandenburg, M.D., Long Beach, Calif.
Floyd E. Dunn, D.O., Kansas City, Mo.
Sidney D. Rothman, D.O., Los Angeles

12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Patient-Doctor Relationship
History Taking and Physical and Neurological Examination
David H. Payne, D.O., Los Angeles

3:00 Diagnostic Aids
Psychological Testing
Electroencephalograph
Electromyograph
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 Problems of Differential Diagnosis
Functional vs. Organic Pathology
Brain Tumor vs. Depressive Reaction
Degenerative Disease, Metabolic Disease, or Neurosis
K. Grosvenor Bailey, D.O., Los Angeles

Wednesday, July 13

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

9:00 Differential Diagnosis
Neurosis vs. Psychosis
K. Grosvenor Bailey, D.O., Los Angeles
10:00 Evaluation of Case Material
Floyd E. Dunn, D.O., Kansas City, Mo.
11:00 Treatment: Osteopathic Manipulation
Floyd E. Dunn, D.O., Kansas City, Mo.
Leonard D. Gluckson, D.O., Los Angeles
Don C. Littlefield, D.O., Long Beach, Calif.
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Treatment:
Psychotherapy for the General Practitioner
Physical Methods
Drug Methods
Surgery, Including Psychosurgery
Floyd E. Dunn, D.O., Kansas City, Mo.
Leonard D. Gluckson, D.O., Los Angeles
Don C. Littlefield, D.O., Long Beach, Calif.
3:00 Depression and Suicide
Leonard D. Gluckson, D.O., Los Angeles
4:00 The Alcoholic Patient
Leonard D. Gluckson, D.O., Los Angeles
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 The Physician's Responsibility to His Community
Edwin F. Peters, Ph.D., Des Moines, Iowa

Thursday, July 14

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

8:00 Hypnosis Seminar
Orientation
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Hypnosis Seminar (cont.)
Technics of Induction
5:00- 7:00 Dinner Period

EVENING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

7:00- 9:00 To be announced

Friday, July 15

MORNING SESSION

Campus of the College of Osteopathic Physicians and Surgeons

8:00 Hypnotic Technic
12:00- 1:00 Luncheon Period

AFTERNOON SESSION

Campus of the College of Osteopathic Physicians and Surgeons

1:00 Hypnotic Technic

RESERVE SPEAKERS

Surgery—A Comparison of European and American Concepts
William E. Merrill, D.O., Seattle
Obstetrical Problems of the Osteopathic General Practitioner in the Suburban Area
H. Roye Rasmusson, D.O., Gresham, Ore.

HOSPITALS THAT WILL PARTICIPATE IN CLINICAL PRESENTATIONS DURING THE CONVENTION INSTRUCTION COURSES

Burbank Hospital, Burbank, Calif.
Doctors Hospital, Los Angeles
Gardena Hospital, Gardena, Calif.
Glendale Community Hospital, Glendale, Calif.
Los Angeles County Osteopathic Hospital, Los Angeles
Magnolia Hospital, Magnolia, Calif.
Maywood Hospital, Maywood, Calif.
Monte Sano Hospital, Los Angeles, Calif.
Park View Hospital, Los Angeles
Rose Hospital, Los Angeles
San Gabriel Hospital, San Gabriel, Calif.
Victory Hospital, North Hollywood, Calif.

PROPOSED SCHEDULE OF FILMS FOR SHOWING AT CONVENTION INSTRUCTION COURSES

1. Surgical Treatment of Arteriovenous Aneurysms by G. H. Pratt, M.D.
   25 minutes—silent—color; Distributed by Davis and Geck, Incorporated, Danbury, Conn.
2. Angina Pectoris by J. E. F. Riseman, M.D.
   90 minutes—sound—color; Distributed by Boston University, Boston
3. Cardiac Arrest by J. C. Owens, M.D., and Henry Swan, M.D
   27 minutes—silent—color; Distributed by University of Colorado, Denver
4. Ligation of the Splenic Artery for Portal Hypertension by Philip Thorek, M.D.
   15 minutes—silent—color; Distributed by Philip Thorek, M.D., Chicago
5. Surgical Treatment of Aneurysms of the Abdominal Aorta by M. E. De Bakey, M.D., and D. A. Cooley, M.D.
   23 minutes—sound—color; Distributed by Davis and Geck, Incorporated, Danbury, Conn.
6. Surgical Treatment of Peripheral Arterial Aneurysms by G. H. Pratt, M.D.
   30 minutes—silent—color; Distributed by St. Vincent's Hospital, New York
7. Electrocardiography by United States Army Medical Department and United States Army Signal Corps
   10 minutes—sound—color; Distributed by United States Army
8. Heart and Lung Preparation by United States Army Medical Department and United States Army Signal Corps
   17 minutes—sound—color; Distributed by United States Army
9. Congenital Malformations of the Heart—Parts I, II, and III by R. F. Rushmer, M.D.
   59 minutes—sound—color; Distributed by University of Washington, Seattle
10. Congenital Pulmonary Stenosis with Intact Interventricular Septum by W. J. Potts, M.D., Stanley Gibson, M.D., and W. L. Riker, M.D.
    30 minutes—silent—color; Distributed by Mervin W. LaRue, Incorporated, Chicago
11. The Heart: Electrokymography, Venous Catheterization and Angiocardiography by W. E. Chamberlain, M.D., Lewis Dexter, M.D., and M. L. Sussman, M.D.
    29 minutes—sound—color; Distributed by D. Searle & Company, Chicago
12. Arteriovenous Fistula with False Sac—Lower Femoral Vessels (Quadruple Ligation and Excision) by United States Army Medical Department and United States Army Signal Corps
    16 minutes—sound—color; Distributed by United States Army
13. Arterial Disorders in the Upper Extremity and their Treatment by Sympathectomy by United States Army Medical Department and United States Army Signal Corps
    31 minutes—sound—color; Distributed by United States Army
14. Management of the Failing Heart by Cornell University Medical College
    40 minutes—sound—color; Distributed by Varick Pharmacal Company, Incorporated, New York
15. Lumbar Sympathectomy Ganglionectomy by J. L. Madden, M.D.
    10 minutes—sound—color; Distributed by Sturgis-Grant Productions, New York
16. Digitalis in the Treatment of Cardiac Failure by Stanford University School of Medicine
    10 minutes—sound—color; Distributed by Sandoz Pharmaceuticals, Hanover, N. J.
17. The Normal Heart Beat Mechanism by Clayton J. Lundy, M.D.
    17 minutes—silent—black and white; Distributed by Sandoz Pharmaceuticals, Hanover, N. J.
18. Extrasystole by Clayton J. Lundy, M.D.
    16 minutes—silent—black and white; Distributed by Sandoz Pharmaceuticals, Hanover, N.J.
19. Auricular Fibrillation by Clayton J. Lundy, M.D.
    9 minutes—silent—black and white; Distributed by Sandoz Pharmaceuticals, Hanover, N.J.
20. Heart Block by Clayton J. Lundy, M.D.
    16 minutes—silent—black and white; Distributed by Sandoz Pharmaceuticals, Hanover, N.J.
21. Influence of Digitalis on the Electrocardiogram by Clayton J. Lundy, M.D.
    17 minutes—silent—black and white; Distributed by Sandoz Pharmaceuticals, Hanover, N.J.
22. Heparin in the Prevention and Treatment of Thrombosis by Upjohn Company
    28 minutes—sound—color; Distributed by Upjohn Company, Kalamazoo, Mich.
23. The Ligation and Stripping Treatment for Varicose Veins by H. O. McPheeters, M. D. and C. V. Kusz, M.D.
    20 minutes—sound—color; Distributed by Davis and Geck, Incorporated, Danbury, Conn.
24. A Cinematographic Study of the Function of the Mitral Valve in Situ by E. S. Hurwitt, M.D., Adrian Kantrowitz, M.D., and Antol Herskovitz, M.D.
    13 minutes—sound—color; Distributed by Medical Audio Visual Institute Services, Chicago
25. Surgical Treatment for Patent Ductus Arteriosus by George H. Humphreys, M.D.
    10 minutes—sound—color; Distributed by Sturgis-Grant Productions, New York
26. Surgical Treatment for Coarctation of the Aorta by Columbia-Presbyterian Medical Center
    20 minutes—sound—color; Distributed by Sturgis-Grant Productions, New York
27. Surgical Treatment for Tetralogy of Fallot by Columbia-Presbyterian Medical Center
    25 minutes—sound—color; Distributed by Sturgis-Grant Productions, New York
28. Side-to-Side Anastomosis of Portal Vein to Vena Cava by Arthur L. Blakemore, M.D.
    10 minutes—silent—color; Distributed by Sturgis-Grant Productions, New York
29. End-to-End Anastomosis of Splenic Vein to Renal Vein by Arthur L. Blakemore, M.D.
    10 minutes—silent—color; Distributed by Sturgis-Grant Productions, New York
30. Lumbar Sympathetic Ganglionectomy (Antero-Lateral Approach—Extra-Peritoneal) by John L. Madden, M.D.
    10 minutes—sound—color; Distributed by Sturgis-Grant Productions, New York

31. The Use of Digitalis in Heart Failure by Wyeth Incorporated
    35 minutes—sound—color; Distributed by Wyeth Incorporated, Philadelphia
32. Surgical Treatment for Congenital Pulmonary Valvular Stenosis by G. H. Humphreys, M.D.
    20 minutes—sound—color; Distributed by Sturgis-Grant Productions, New York
33. Anemia by William Dameshek, M.D.
    25 minutes—sound—color; Distributed by E. R. Squibb and Sons, New York
34. Pediatric Anesthesia by Digby Leigh, M.D.
    30 minutes—silent—color; Distributed by E. R. Squibb and Sons, New York
35. Curare in Barbiturate-Oxygen Anesthesia by T. C. Davison, M.D., and A. H. Letton, M.D.
    20 minutes—sound—color; Distributed by E. R. Squibb and Sons, New York
36. Endotracheal Anesthesia by Charles McCuskey, M.D.
    20 minutes—sound—color; Distributed by E. R. Squibb and Sons, New York
37. Pudendal Block with Demerol and Intracaine by Lees M. Schadel, M.D.
    10 minutes—sound—color; Distributed by E. R. Squibb and Sons, New York
38. Curare-Intocostrin, d-Tubocurarine Chloride, Mecostrin by John B. Dillon, M.D.
    25 minutes—sound—color; Distributed by E. R. Squibb and Sons, New York
39. Post-Anesthesia Rooms by Scott M. Smith, M.D.
    25 minutes—sound—color; Distributed by E. R. Squibb and Sons, New York
40. Infiltration Anesthesia in the First Stage of Labor by A. A. Abrams, M.D.
    20 minutes—sound—color; Distributed by E. R. Squibb and Sons, New York
41. Refrigeration Anesthesia (in a Leg Amputation) by F. M. Allen, M.D., and L. W. Crossman, M.D.
    30 minutes—silent—color; Distributed by E. R. Squibb and Sons, New York

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Convention Teaching Sessions

Monday, July 18

MORNING SESSION

Pacific Ballroom, Hotel Statler

8:10 Visual Education Film: Procedures in the Diagnosis of Cardiovascular Diseases by University of Southern California, Los Angeles
Patent Ductus Arteriosus by E. R. Squibb & Sons, New York

9:00 Call to order
John W. Mulford, D.O., Cincinnati
PRESIDENT, AMERICAN OSTEOPATHIC ASSOCIATION
Invocation
Introduction of Program Chairman
John W. Mulford, D.O., Cincinnati
PRESIDENT, AMERICAN OSTEOPATHIC ASSOCIATION

9:15 Keynote Address—Should We Be Interested in World Health?
Wilton L. Halverson, M.D., Los Angeles
ASSOCIATE DEAN, SCHOOL OF PUBLIC HEALTH, UNIVERSITY OF CALIFORNIA

10:00 Pathologic Manifestations of the Arteriosclerotic State
Norman W. Arends, D.O., Huntington Woods, Mich.

10:30 The Role of the General Practitioner in the Diagnosis and Treatment of Acute Cerebral Thrombosis and Hemorrhage
John V. Fiore, D.O., Los Angeles

10:50 Cerebral Trauma: Diagnostic Criteria for the Early Detection and Treatment of Intracranial Hemorrhage
Randall J. Chapman, D.O., Burbank, Calif.

11:10 Manipulative and Rehabilitative Care of the Cerebrovascular Cripple
Clayton H. Morgan, D.O., Kansas City, Mo.

11:40 Psychotherapeutic Considerations in the Prevention and Control of Vascular Headaches
R. J. Tuche, D.O., Oakland, Calif.

12:00 Noon Recess

AFTERNOON SESSION I—LECTURE SESSION

Pacific Ballroom, Hotel Statler

2:00 Criteria for the Diagnosis of Heart Disease
Glennard E. Lahrson, D.O., Oakland, Calif.

2:30 Acute Rheumatic Fever: Modern Methods of Diagnosis and Treatment
Robert S. Sedar, D.O., Denver, Colo.

3:00 Diagnosis and Treatment of the Hemorrhagic Diseases
S. J. Aquilla, Van Nuys, Calif.

3:30 Diagnostic and Medical Therapeutic Considerations in Diseases of the Portal System
Stuart F. Harkness, D.O., Des Moines, Iowa

4:00 Peripheral Vascular Complications of Pregnancy
Donald T. Sheldon, D.O., Los Angeles

4:30 Newer Concepts in Anticoagulation Therapy
A. Verne Jackson, D.O., Forest Grove, Ore.

5:00 Adjournment

AFTERNOON SESSION II—MANIPULATIVE SESSION

Auditorium and Campus of the College of Osteopathic Physicians and Surgeons

Director of Sessions: Robert C. Ruenitz, D.O., Los Angeles

2:00 The Role of the Specialist in Physical Medicine and Rehabilitation in the Practice of Osteopathic Medicine of Today
William W. W. Pritchard, D.O., Pasadena, Calif.

2:40 Discussion and Demonstrations
William W. W. Pritchard, D.O., Pasadena, Calif.

3:00 The Physical Medicine and Rehabilitation Program of the Los Angeles College of Osteopathic Physicians and Surgeons
J. M. Andrews, D.O., Los Angeles

3:40 Discussion and Demonstration of Technics of the Physical Therapist
J. M. Andrews, D.O., Los Angeles

4:00 The Value of Occupational Therapy and Manipulation in the Rehabilitation of the Cerebral Palsied Victim
Beryl E. Arbuckle, D.O., Philadelphia

4:40 Discussion and Technic Demonstration
Beryl E. Arbuckle, D.O., Philadelphia
William W. W. Pritchard, D.O., Pasadena, Calif.
J. M. Andrews, D.O., Los Angeles

2:00-5:00 Clinical demonstrations of occupational therapy, physical medicine, and rehabilitative technics, at the College of Osteopathic Physicians and Surgeons Rehabilitation Center, and the Los Angeles County Osteopathic Hospital

Note: Registered physicians will be conducted through these departments by groups each half hour. Tickets must be obtained at Registration Desk.

Tuesday, July 19

MORNING SESSION

Pacific Ballroom, Hotel Statler

8:10 Visual Education Film: Mitral Commissurotomy by Bailey Thoracic Clinic, Philadelphia
Aortic Commissurotomy by Bailey Thoracic Clinic, Philadelphia

9:00 Functional Cardiac Problems—A Discussion of the Rationale of a Manipulative Approach for their Therapeutic Control
S. V. Robuck, D.O., Chicago

9:30 The Cardiac Cripple — Differential Diagnosis and Therapeutic Control of the Acquired Cardiac Lesions
Harold J. Strick, D.O., Los Angeles

10:00 The Clinical Significance of the Atherogenic Index
John W. Gofman, M.D., Berkeley, Calif.

10:45 The Importance of Research to Osteopathic Institutions and Osteopathic Practices
Moderator: W. Ballentine Henley, LL.D., Los Angeles
Discussions by:
Alexander Levitt, D.O., Brooklyn
Lawrence R. Morehouse, Ph.D., Los Angeles
Irvin M. Korr, Ph.D., Kirksville, Mo.

12:15 Noon Recess

AFTERNOON SESSION I—LECTURE SESSION

Pacific Ballroom, Hotel Statler

2:00 Coronary Artery Disease—Diagnostic and Therapeutic Considerations with Special Emphasis on the Role of the General Practitioner in Early Diagnosis and Treatment
J. Claver Cargill, D.O., Burbank, Calif.

2:30 Cardiac Murmurs—A Discussion of the Significant Normal and Abnormal Cardiac Sounds and Their Time Relationship to the Cardiac Cycle
Munish Feinberg, D.O., Los Angeles

3:00 Visual Education Film: The Damaged Cardiac Valve — An original animated sound and color film depicting the common cardiac valvular conditions together with sound track of cardiac murmurs as recorded through the electronic stethoscope
Munish Feinberg, D.O., Los Angeles
Robert M. Loveland, D.O., Los Angeles

3:45 Congenital Heart Disease in Infants and Children— Pathologic and Therapeutic Considerations
Wayne G. Peyton, D.O., Sacramento, Calif.

4:30 Cardiac Arrest — Medicolegal and Therapeutic Responsibilities of Anesthetists, Surgeons, and General Practitioners
J. Donald Sheets, D.O., Highland Park, Mich.

5:00 Adjournment

AFTERNOON SESSION II—MANIPULATIVE SESSION

Sierra Room, Hotel Statler

Director of Sessions: Bruce S. Collins, D.O., Santa Monica, Calif.

2:30 Physical Medicine and Manipulation in the Management of Arteriosclerosis
William T. Sechrist, D.O., Los Angeles

3:15 Whiplash Injuries to the Cervicodorsal Area with Special Reference to Diagnosis and Application of Manipulative Therapy
Troy L. McHenry, D.O., Los Angeles

4:00 Differential Diagnosis and Management of Low-Back Problems with Emphasis on Manipulative Therapy
Silas Williams, D.O., Santa Paula, Calif.

4:45 Discussion and Demonstration of Technics
Troy L. McHenry, D.O., Los Angeles
Silas Williams, D.O., Santa Paula, Calif.
William T. Sechrist, D.O., Los Angeles

5:15 Adjournment

Wednesday, July 20

MORNING SESSION

Pacific Ballroom, Hotel Statler

8:10 Visual Education Film: Pheochromocytoma, a Cause of Hypertension — Diagnosis and Treatment by Ciba Pharmaceutical Products Incorporated, Summit, N. J.

Arteriovenous Fistula with False Sac — Lower Femoral Vessels (Quadruple Ligation and Excision) by United States Army Medical Department and United States Army Medical Corps

Symposium on Essential Hypertension
Moderator: Robert K. Schieffer, D.O., San Diego, Calif.

9:00 The Role of the Autonomic Nervous System and the Kidney
Donald E. Pinder, D.O., Los Angeles

9:20 Differential Diagnosis
J. F. DePetris, D.O., Dallas, Tex.

9:40 Principles of Manipulative Management
Richard Sayre Koch, D.O., Olympia, Wash.

10:00 Principles of Medical Management
Maxwell R. Brothers, D.O., Los Angeles

10:20 Principles of Surgical Management
Karl Albaeck, D.O., Burbank, Calif.

10:40 Question and Answer Period

11:00 Andrew Taylor Still Memorial Lecture — Our Great and Noble Heritage
T. L. Northup, D.O., Morristown, N. J.

12:00 Noon Recess

AFTERNOON SESSIONS I AND II (COMBINED)

Auditorium and Campus of the Los Angeles College of Osteopathic Physicians and Surgeons

2:00 Diagnostic and Therapeutic Implications of Radioactive Isotopes
M. H. Simmers, D.O., Pasadena, Calif.

2:30 Demonstration of the Clinical Applications of Radioactive Isotopes
M. H. Simmers, D.O., Pasadena, Calif.

3:00 Poliomyelitis: The Role of the Electromyograph and Its Application in Treatment, Including Clinical Demonstrations
John A. Schuck, D.O., Los Angeles

4:00 Cardiovascular Research Program of the College of Osteopathic Physicians and Surgeons
Delmar J. Daniel, D.O., Burbank, Calif.

4:30 Visual Education Film: Application Prosthetics
Delmar J. Daniel, D.O., Burbank, Calif.

5:00 Adjournment

Thursday, July 21

MORNING SESSION

Pacific Ballroom, Hotel Statler

8:10 Visual Education Film: Vascular Injuries (color) by United States Army Medical Department and United States Army Medical Corps

Symposium on Arteriosclerosis

Moderator: Glennard E. Lahrson, D.O., Oakland, Calif.

9:00 Symptomatology — Early and Late — of Arterial Aging of the Brain, Heart, Kidneys, and Extremities
Robert K. Schieffer, D.O., San Diego, Calif.

9:45 Retinal Manifestations and Vascular Disease
Preston J. Stack, D.O., Los Angeles

10:15 Medical Prophylaxis and Therapy in Cerebral, Cardiac, Renal, and Peripheral Arteriosclerosis
Neil R. Kitchen, D.O., Detroit

10:45 Indications and Types of Anesthesia for the Arteriosclerotic Patient
Burnham Brooke, D.O., Portland, Ore.

11:15 The Surgical Management of Arteriosclerosis Obliterans
William K. Eaton., Jr., D.O., Sacramento, Calif.

11:45 Questions and Answers

12:15 Noon Recess

AFTERNOON SESSION I

Pacific Ballroom, Hotel Statler

2:00 Diagnostic and Therapeutic Management of Varicose Veins
Walter E. Mattocks, D.O., Long Beach, Calif.
Wallin W. King, D.O., Los Angeles

2:45 Chronic Ulcerations of the Lower Extremities — Differential Diagnosis and Treatment
John A. Costello, D.O., Los Angeles
Joseph O. Costello, D.O., Los Angeles

3:30 Embolic Catastrophes Involving the Arterial and Venous Systems (with Special Emphasis on the Criteria for Early Recognition by the General Practitioner and Accepted Methods of Treatment)
LaMoyne W. Hickman, D.O., Los Angeles
Roger B. Anderson, D.O., Manning, Iowa

4:15 The Restoration of Arterial Continuity by Direct Surgical Attack
W. Donald Baker, D.O., Los Angeles
William T. Barrows, D.O., Oakland, Calif.

5:00 Adjournment

AFTERNOON SESSION II—MANIPULATIVE SESSION

Sierra Room, Hotel Statler

Director of Session: Robert C. Ruenitz, D.O., Los Angeles

2:00 Neuromuscular Mechanisms
L. R. Morehouse, Ph.D., Los Angeles

2:45 Audiovisual Film: Mobilization of the Human Body
Harvey Billig, M.D., Los Angeles

3:15 Indications and Contraindications for Manipulative Management of Rheumatoid Problems
Harvey Billig, M.D., Los Angeles

4:00 Diagnostic and Manipulative Considerations of Shoulder Disabilities
Elmer S. Clark, D.O., Long Beach, Calif.

4:30 Audiovisual Film: Demonstration of Shoulder Technics
Elmer S. Clark, D.O., Long Beach, Calif.

5:00 Adjournment

Friday, July 22

MORNING SESSION

Sierra Room, Hotel Statler

Round Table Discussion on Geriatric Problems

Moderator: Harold D. Meyer, D.O., Algona, Iowa

9:00 Social and Economic Aspects of the Aging
Mr. Louis Kuplan, Executive Secretary, Inter-Departmental Coordinating Committee on Aging, State of California, Sacramento, Calif.

9:20 The Responsibilities of the General Practitioner in the Care of Geriatric Cases
Robert D. McCullough, D.O., Tulsa, Okla.

9:40 Our Surgical Responsibilities to the Aged
Paul Atterberry, D.O., Milwaukee

10:00 Manipulative Problems Encountered in a Geriatric Practice
Robert B. Thomas, D.O., Huntington, W. Va.

10:40 Orthopedic Tragedies in Geriatric Cases
James M. Eaton, D.O., Philadelphia

11:00 Urologic Problems and Their Management in the Geriatric Patient
George F. Scouten, D.O., Long Beach, Calif.

11:20 Neuropsychiatric Considerations Peculiar to the Aged
K. Grosvenor Bailey, D.O., Los Angeles

11:40 Round Table Discussion (or written questions submitted to panel)

12:30 Adjournment

Note: Forms will be made available at the time of registration and throughout the convention for written questions to be submitted to the Geriatric panel.

Schedule for Official Family

BOARD OF TRUSTEES
July 12 (only)—Mission Room
July 13-22—Los Angeles Room
HOUSE OF DELEGATES
July 17-22—Golden State Room
Statler Hotel
Los Angeles

Tuesday, July 12
10:00-10:10 Executive Committee
10:10- 1:00 Board of Trustees
2:00- 6:00 Board of Trustees
*7:30-10:00 Board of Trustees

Wednesday, July 13
9:00- 1:00 Board of Trustees
2:00- 6:00 Board of Trustees
*7:30-10:00 Board of Trustees

Thursday, July 14
9:00- 1:00 Board of Trustees
2:00- 6:00 Board of Trustees
*7:30-10:00 Board of Trustees

Friday, July 15
9:00- 1:00 Board of Trustees
2:00- 6:00 Board of Trustees
*7:30-10:00 Board of Trustees

*Evening meetings will be called if necessity therefor is apparent.

Saturday, July 16
9:00- 1:00 Board of Trustees
2:00- 5:00 Board of Trustees

Sunday, July 17
9:00-12:00 Board of Trustees
12:00- 1:00 Registration of Delegates
1:00- 5:30 House of Delegates
7:00-10:30 House of Delegates

Monday, July 18
9:00-12:00 House of Delegates
1:30- 5:30 House of Delegates
7:00 President's Banquet, Reception, and Ball — Hollywood Palladium

Tuesday, July 19
8:00-10:00 Board of Trustees
10:00-12:00 House of Delegates
2:00- 6:00 House of Delegates
7:00 Fraternity and Sorority Banquets

Wednesday, July 20
8:00-11:00 Board of Trustees
11:00-12:00 A. T. Still Memorial Address—Pacific Ballroom (Officers and Trustees attend in a body)
2:00- 6:00 House of Delegates
7:00 Alumni Banquets

Thursday, July 21
8:00-10:00 Board of Trustees
10:00-12:00 House of Delegates
2:00- 5:30 House of Delegates
7:00 Inaugural Banquet and Installation of Officers — Hollywood Palladium

Friday, July 22
8:00-10:00 House of Delegates

Bureau and Committee Meetings

COMMITTEE ON AUDIO-VISUAL EDUCATION AND INFORMATION
July 12, 7:30 p.m., Dinner and Meeting—Buffalo Room, Hotel Statler
Chairman—George W. Northup, D.O., Livingston, N. J.
Director—Mr. T. F. Lindgren, Chicago

COMMITTEE ON ACCREDITATION OF POSTGRADUATE TRAINING
July 14, 15, 16, 9:00 a.m.-10:00 p.m.—New York Room, Hotel Statler
Chairman—William Baldwin, Jr., D.O., Philadelphia
Secretary—Robert A. Steen, D.O., Chicago

CONVENTION CITY COMMITTEE
July 16, 7:30 a.m., Breakfast—Foy Room, Hotel Statler
Chairman—E. H. McKenna, D.O., Muskegon Heights, Mich.

1956 PROGRAM COMMITTEE
July 18, 12:30 p.m., Luncheon—Mission Room, Hotel Statler
Chairman—Myron C. Beal, D.O., Rochester, N. Y.

EDITORIAL LUNCHEON
July 19, 12:30 p.m., Luncheon—Conference Room No. 3, Biltmore Hotel
Co-Chairmen—Raymond P. Keesecker, D.O., Chicago
Mrs. Katherine Becker, Chicago

OSTEOPATHIC PROGRESS FUND COMMITTEE
July 13, 7:30 p.m., Dinner—New York Room, Hotel Statler
Chairman—Galen S. Young, D.O., Chester, Pa.
Director—Mr. Lewis F. Chapman, Chicago

COMMITTEE ON PROFESSIONAL LIABILITY INSURANCE
July 17, 1:00-5:00 p.m.—Mission Room, Hotel Statler
Chairman—Forest J. Grunigen, D.O., Los Angeles

Specialty Boards

ADVISORY BOARD FOR OSTEOPATHIC SPECIALISTS
July 17, 18, 19, 9:00 a.m.-10:00 p.m.—New York Room, Hotel Statler
Chairman—Earl E. Congdon, D.O., Flint, Mich.
Secretary—Robert A. Steen, D.O., Chicago

AMERICAN OSTEOPATHIC BOARD OF INTERNAL MEDICINE
July 16, 10:00 a.m.-6:00 p.m.—Conference Room No. 6, Biltmore Hotel
Chairman—H. Earle Beasley, D.O., Boston
Secretary-Treasurer—Glennard E. Lahrson, D.O., Oakland, Calif.

AMERICAN OSTEOPATHIC BOARD OF NEUROLOGY AND PSYCHIATRY
July 14, 9:00 a.m.-6:00 p.m.—Conference Room No. 3, Biltmore Hotel
Chairman—K. Grosvenor Bailey, D.O., Los Angeles
Secretary-Treasurer—Thomas J. Meyers, D.O., Pasadena, Calif.

AMERICAN OSTEOPATHIC BOARD OF OBSTETRICS AND GYNECOLOGY
July 16, 9:00 a.m.-5:00 p.m.—Conference Room No. 5, Biltmore Hotel
Chairman—Dorothy J. Marsh, D.O., Los Angeles
Secretary-Treasurer—Jacquelin Bryson, Denver

AMERICAN OSTEOPATHIC BOARD OF PATHOLOGY
July 15, 9:00 a.m.-5:00 p.m.—Examinations at Campus of College of Osteopathic Physicians and Surgeons, Los Angeles
7:00 p.m., Dinner—Perrino's Restaurant
Chairman—Robert P. Morhardt, D.O., South Pasadena, Calif.
Secretary-Treasurer—Norman W. Arends, D.O., Huntington Woods, Mich.

AMERICAN OSTEOPATHIC BOARD OF PEDIATRICS

July 13, 14, 15, 9:00 a.m.-5:00 p.m.—Conference Room No. 5, Biltmore Hotel

Chairman—William S. Spaeth, D.O., Drexel Hill, Pa.
Secretary-Treasurer—H. Mayer Dubin, D.O., Los Angeles

AMERICAN OSTEOPATHIC BOARD OF PROCTOLOGY

July 16, 9:00 a.m.-5:00 p.m.—Conference Room No. 3, Biltmore Hotel

Chairman—Carl S. Stillman, Jr., D.O., San Diego, Calif.
Secretary-Treasurer—C. M. Noll, D.O., Denver

AMERICAN OSTEOPATHIC BOARD OF PHYSICAL MEDICINE AND REHABILITATION

July 15, 16, 9:00 a.m.-5:00 p.m.—Cleveland Room, Hotel Statler

Chairman—W. W. W. Pritchard, D.O., Pasadena, Calif.
Secretary-Treasurer—Robert C. Ruenitz, D.O., Los Angeles

AMERICAN OSTEOPATHIC BOARD OF SURGERY

July 13, 14, 15, 8:00 a.m-10:00 p.m.—Buffalo Room, Hotel Statler

Chairman—James M. Eaton, D.O., Philadelphia
Secretary-Treasurer—Arthur M. Flack, D.O., Philadelphia

Allied Organizations

ACADEMY OF APPLIED OSTEOPATHY

July 19, 1:30 p.m., Executive Board Meeting—Space to be announced
July 22, 9:00-11:00 a.m., Board of Governors—Mission Room, Hotel Statler
12:30 p.m., Luncheon—Sierra Room, Hotel Statler
2:00-5:00 p.m., 7:00-10:30 p.m.—Lecture Sessions—Sierra Room, Hotel Statler
July 23, 9:00 a.m.-5:00 p.m., Lecture Sessions—Sierra Room, Hotel Statler
12:15 p.m., Luncheon for Board of Governors—Cleveland Room, Hotel Statler

President—William A. Ellis, D.O., Grand Rapids, Mich.
Secretary—Margaret W. Barnes, D.O., Carmel, Calif.

Friday, July 22

Afternoon

12:30 Academy Luncheon
Address of the President
William A. Ellis, D.O., Grand Rapids, Mich.
2:00 Cardiac Anatomy
Scott B. Wisner, D.O., Seattle
2:30 Diagnosis of Cardiac Problems and Significance of Functional Testing
R. MacFarlane Tilley, D.O., Kirksville, Mo.
3:15 Annual Business Meeting
3:45 Incoming President's Address
C. Raymond Nelson, D.O., San Antonio, Tex.
4:00 Osteopathic Cardiology
Perrin T. Wilson, D.O., Cambridge, Mass.
5:00 Adjourn for the afternoon

Evening

7:30 Clinical Aspects of Osteopathic Research
J. S. Denslow, D.O., Kirksville, Mo.
8:00 The Industrial Accident
George M. McCole, D.O., Great Falls, Mont.
8:45 Dr. Still's Swing and Other Technics
Asa Willard, D.O., Missoula, Mont.
9:30 Question period and additional demonstration time, featuring the speakers of the day
10:15 Adjourn for the evening

Saturday, July 23

Morning

9:00 Eighth Annual Academy Lecture—Osteopathy in the Field of Preventive Medicine
Thomas L. Northup, D.O., Morristown, N. J.
10:00 A Functional Technic—Panel Presentation by the Puget Sound Academy of Applied Osteopathy
H. V. Hoover, D.O., Coordinator, Tacoma, Wash.
Assumptions Necessary to Understand This Technic
Mary Alice Hoover, D.O., Tacoma, Wash.
The Experimental Method of Learning
M. D. Young, D.O., Seattle
Experiments to Determine Nature of This Functional Technic with Audience Participation—Supervised by the Panel

Afternoon

12:15 Luncheon for Academy Officers, Trustees, and Governors
2:00 Continuation of A Functional Technic—Panel Presentation by Puget Sound Academy of Applied Osteopathy
Complicated Lesions
J. Lowell Kinslow, D.O., Seattle
Patterns of Lesions
Thomas C. Herren, D.O., Kelso, Wash.
Theory of This Functional Technic
Mary E. Gillies, D.O., Seattle
Comparison of Functional with Structural Technics
E. E. Lacroix, D.O., Seattle
Application of This Functional Technic
J. C. Hendrick, D.O., Bremerton, Wash.
Summary
H. V. Hoover, D.O., Tacoma, Wash.
Practice by audience, supervised by Panel members. Questions directed to Panel members and Panel discussion
4:30 Adjourn convention

RESERVE SPEAKERS

Clyde W. Dalrymple, D.O., Little Rock, Ark.
Harry L. Davis, D.O., Walla Walla, Wash.
Allan A. Eggleston, D.O., Montreal, Canada

Osteopathic Cranial Association

July 23, 6:00 p.m., Banquet—Los Angeles Room, Hotel Statler
8:00-10:00 p.m., Meeting—Los Angeles Room, Hotel Statler
July 24, 9:00 a.m.-5:00 p.m., Lecture Session—Los Angeles Room, Hotel Statler

President—Samuel H. Hitch, D.O., Lubbock, Tex.
Executive Secretary—Kenneth E. Little, D.O., Kansas City, Mo.
Program Chairman—William C. Rankin, D.O., Marietta, Ohio

AMERICAN ASSOCIATION OF OSTEOPATHIC COLLEGES

July 15, 16, 9:00 a.m.-5:00 p.m.—Mission Room, Hotel Statler

President—Mr. J. M. Peach, Kansas City, Mo.
Secretary-Treasurer—J. S. Denslow, D.O., Kirksville, Mo.

AMERICAN ASSOCIATION OF OSTEOPATHIC EXAMINERS

July 20, 12:15 p.m., Luncheon—Cleveland Room, Hotel Statler

President—Marion E. Coy, D.O., Jackson, Tenn.
Secretary-Treasurer—John H. Warren, D.O., Philadelphia

AMERICAN COLLEGE OF GENERAL PRACTITIONERS IN OSTEOPATHIC MEDICINE AND SURGERY

July 22, 2:30-5:00 p.m., Meeting—Pacific Ballroom, Hotel Statler

8:30 p.m., Banquet—Pacific Ballroom, Hotel Statler

President—Frank York Lee, D.O., Los Angeles
Secretary—Alfred J. Schramm, D.O., Los Angeles

AMERICAN OSTEOPATHIC ACADEMY OF PHYSICAL MEDICINE AND REHABILITATION

July 23, 8:00 a.m.-5:00 p.m., Lecture Sessions—Los Angeles Room, Hotel Statler

12:30 p.m., Luncheon—New York Room, Hotel Statler

President—W. Irvin Harner, D.O., Whittier, Calif.
Secretary-Treasurer—John A. Schuck, D.O., Los Angeles

Saturday, July 23

Morning

8:00 Business Meeting

10:00 Osteopathic Principles Applied in the Treatment of Arthritis
E. C. Andrews, D.O., Ottawa, Ill.

11:00 Neurophysical Aspects of Neuromuscular Diseases
Karl Albaeck, D.O., Burbank, Calif.

Afternoon

12:30 Luncheon

2:00 Physical Medicine in the Rehabilitation of the Orthopedic Patient
C. Robert Starks, D.O., Denver

3:00 Uses of Physical Medicine in the Care of Obstetrical Patients
Richard E. Eby, D.O., Pomona, Calif.

4:00 Present-Day Physical Medicine Modalities and Their Therapeutic Application
Harvey S. Taylor, D.O., Los Angeles

5:00 Closing minutes and adjournment of the Convention

AMERICAN OSTEOPATHIC COLLEGE OF PATHOLOGY

July 14, 10:00 a.m.-5:00 p.m., Lecture Sessions—Conference Room No. 7, Biltmore Hotel

6:00 p.m., Banquet—Location to be announced

8:00-10:00 p.m., Business Meeting—Conference Room No. 7, Biltmore Hotel

July 15, 9:00 a.m.-5:00 p.m., Visitations to Hospitals in Los Angeles Area

Chairman Pro Tem—George E. Himes, D.O., Flint, Mich.

Thursday, July 14

Morning

10-10:50 Serum Proteins
George E. Himes, D.O., Flint, Mich.

11-11:50 Syndrome of Ovarian Pain and Insufficiency
William J. Loos, D.O., Detroit

Afternoon

2- 2:50 Choriocarcinoma
W. Harriet Davis, D.O., Burbank, Calif.

3- 3:50 Long-Range Results of Pelvic Surgery
Ray McClure, D.O., Los Angeles

4- 4:50 Death Mechanisms and Causes
Otterbein Dressler, D.O., Highland Park, Mich.

Evening

6- 8:00 Dinner for Members and Wives

8-10:00 Meeting for Business of Association

Friday, July 15

9:00 a.m.-5:00 p.m. Visitation to Los Angeles County Osteopathic Hospital, Burbank Hospital, and San Gabriel Valley Hospital

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AMERICAN OSTEOPATHIC COLLEGE OF GERIATRICS

July 20, 21, 2:00-5:00 p.m., Business Meetings—Washington Room, Hotel Statler

July 22, 2:00-6:00 p.m., Lecture Sessions—Cleveland Room, Hotel Statler

President—E. H. McKenna, D.O., Muskegon Heights, Mich.
Secretary-Treasurer—J. K. Johnson, Jr., D.O., Jefferson, Iowa

Friday, July 22

Afternoon

The Aging Process, A Medical and Sociological Problem—Presidential Address
E. H. McKenna, D.O., Muskegon Heights, Mich.

The Degenerative Processes, A Problem of the Aging Patient
Alexander Levitt, D.O., Brooklyn

Structural Problems in the Aging Patient
George W. Northup, D.O., Livingston, N. J.

Cardiovascular-Renal Problems in the Aging Patient
Claude Van Andel, D.O., Muskegon, Mich.

Surgical Problems of the Geriatric Patient
H. Dale Pearson, D.O., Erie, Pa.

AMERICAN OSTEOPATHIC GOLF ASSOCIATION

July 19, 12:00 noon, Luncheon—Fox Hills Golf and Country Club

1:00 p.m., Tee-off Time—Fox Hills Golf and Country Club

President—Harry P. Stimson, D.O., Highland Park, Mich.
Secretary-Treasurer—H. L. Gulden, D.O., Ames, Iowa
Program Chairman—Robert C. Ruenitz, D.O., Los Angeles

AMERICAN OSTEOPATHIC SOCIETY FOR THE STUDY AND CONTROL OF RHEUMATIC DISEASES

July 17, 9:00 a.m.-1:00 p.m., Lecture Session—Cleveland Room, Hotel Statler

President—L. P. Ramsdell, D.O., La Porte, Ind.
Secretary-Treasurer—E. C. Andrews, D.O., Ottawa, Ill.
Program Chairman—Wilbur V. Cole, D.O., Kansas City, Mo.

ASSOCIATION OF OSTEOPATHIC PUBLICATIONS

July 16, 12:30 p.m., Luncheon and Meeting with the Society of Divisional Secretaries—Conference Room No. 8, Biltmore Hotel

President—Mr. Paul Adams, Jefferson City, Mo.
Secretary-Treasurer—Miss Josephine Seyl, Chicago
Program Chairman—Mr. Wendell Fuller, Des Moines, Iowa

Saturday, July 16

12:30 Luncheon

2:00 Osteopathic Publications—Their Functions and Effectiveness

Moderator: Mr. D. David Darland, Chicago
DIRECTOR, DIVISION OF PUBLIC AND PROFESSIONAL WELFARE, AMERICAN OSTEOPATHIC ASSOCIATION

The Purpose of Osteopathic Publications from the Standpoint of the A.O.A.

Raymond P. Keesecker, D.O., Chicago
EDITOR, AMERICAN OSTEOPATHIC ASSOCIATION

Your Publication—Past or Present
Mr. Richard Hoffman, Los Angeles
LOS ANGELES CITY COLLEGE PRINTING DEPARTMENT

Divisional Society and Auxiliary Publications
Mr. David J. Rodgers, Los Angeles
ASSISTANT EXECUTIVE SECRETARY, CALIFORNIA OSTEOPATHIC ASSOCIATION

College Publications
Mr. Clifton Cornwell, Kirksville, Mo.
EDITOR, *The Stilletto;* MANAGING EDITOR, *Journal of Osteopathy,* KIRKSVILLE COLLEGE OF OSTEOPATHY AND SURGERY

Hospital Publications
Mr. William S. Konold, Columbus, Ohio
EXECUTIVE SECRETARY AND EDITOR, OHIO OSTEOPATHIC ASSOCIATION OF PHYSICIANS AND SURGEONS; EDITOR, *Journal of the Columbus Clinical Group;* FORMER EXECUTIVE SECRETARY, AMERICAN OSTEOPATHIC HOSPITAL ASSOCIATION

4:30 Business Meeting of the Association of Osteopathic Publications

AUXILIARY TO THE AMERICAN OSTEOPATHIC ASSOCIATION

July 17-23—Biltmore Hotel

President—Mrs. Paul van B. Allen, Indianapolis, Ind.
President-Elect—Mrs. George Cozma, Cleveland
First Vice President—Mrs. E. W. Egle, Clayton, Mo.
Second Vice President—Mrs. W. H. Bethune, Grand Rapids, Mich.
Treasurer—Mrs. Munroe H. Kneeland, Liberal, Mo.
Recording Secretary—Mrs. Henry L. McDowell, Norwalk, Calif.
Corresponding Secretary—Mrs. J. Wesley Elbert, Petersburg, Ind.

Sunday, July 17
Afternoon
Conference Room No. 7
1:30- 5:30 Executive Board Meeting

Monday, July 18
Morning
8:00-10:00 Student Wives Council, Breakfast—Conference Room No. 3
Guild Committee Meeting, Breakfast—Conference Room No. 7
10:00-12:00 Formal Opening—Music Room

Afternoon
3:00- 5:00 A.O.A. Social Event

Tuesday, July 19
Morning
Music Room
8:00- 9:00 Workshop
9:00-11:00 House of Delegates
11:00-12:00 Address—The Educational Profile of Osteopathy
Edwin F. Peters, Jr., Ph.D., Des Moines, Iowa
PRESIDENT, DES MOINES STILL COLLEGE OF OSTEOPATHY AND SURGERY

Afternoon
Music Room
1:30- 5:00 House of Delegates

Wednesday, July 20
Morning
Music Room
8:00- 9:00 Workshop
9:00-11:00 House of Delegates
Pacific Ballroom, Hotel Statler
11:00-12:00 Andrew Taylor Still Memorial Lecture

Afternoon
Conference Room No. 7
12:30- 3:00 State President's Luncheon
Mrs. George Cozma, Cleveland, President-Elect, presiding
Conference Room No. 3
3:00- 5:00 Meeting of Newly Elected Officers

Thursday, July 21
Morning
Music Room
9:00-12:00 House of Delegates

Rendezvous Room
1:00- 3:00 Luncheon Honoring Newly Elected Officers
Mrs. E. W. Egle, Clayton, Mo., First Vice President and Program Chairman, presiding
Address
Mrs. George Cozma, Cleveland, Incoming President
Entertainment

Friday, July 22
Morning
Conference Room No. 7
9:00-12:00 Committee Meetings

Afternoon
Conference Room No. 7
1:00- 5:00 Executive Board Meeting

Saturday, July 23
Morning
Conference Room No. 7
9:00-12:00 Executive Board Meeting

GAVEL CLUB

July 18, 7:30 a.m., Breakfast—Washington Room, Hotel Statler

President—R. M. Tilley, D.O., Kirksville, Mo.
Secretary-Treasurer—Robert B. Thomas, D.O., Huntington, W. Va.

LOS ANGELES PROCTOLOGICAL SOCIETY

July 19, 8:00 p.m., Dinner honoring Frank D. Stanton, D.O., Boston—Conference Room No. 8, Biltmore Hotel
Banquet Chairman—Philip F. Spooner, D.O., Los Angeles

NATIONAL BOARD OF EXAMINERS FOR OSTEOPATHIC PHYSICIANS AND SURGEONS

July 16, 17, 19, 10:00 a.m.-5:00 p.m.—Washington Room, Hotel Statler

President—S. V. Robuck, D.O., Chicago
Secretary-Treasurer—Paul van B. Allen, D.O., Indianapolis

THE OSTEOPATHIC FOUNDATION

Date and space to be announced

President—John W. Mulford, D.O., Cincinnati
Secretary—R. C. McCaughan, D.O., Chicago

OSTEOPATHIC LIBRARIES ASSOCIATION

July 14, 9:00 a.m.-12:00 noon, Business Meeting—Conference Room No. 6, Biltmore Hotel
2:00-3:30 p.m., Tour of Library—College of Osteopathic Physicians and Surgeons
3:30-4:00 p.m., Address
W. Ballentine Henley, LL.D., Los Angeles
4:00-5:00 p.m., Tea—College of Osteopathic Physicians and Surgeons
July 15, 9:00 a.m.-12:00 noon, Tour of Libraries—Los Angeles Area
2:00-3:30 p.m., Round Table Discussion: Technical Process in Osteopathic Libraries
Mrs. Frances H. Beisel, Philadelphia, Moderator
3:30-5:00 p.m., Round Table Discussion: Better Coordination of Activities Among Osteopathic Libraries
Miss Lorraine Lloyd, Chicago, Moderator

President—Mr. Francis M. Walter, Kirksville, Mo.
Secretary-Treasurer—Miss Margaret Cressaty, Los Angeles

OSTEOPATHIC TRUST

Date and space to be announced

President—George W. Riley, D.O., New York (Deceased)
Secretary—R. C. McCaughan, D.O., Chicago

OSTEOPATHIC VOCATIONAL GROUP OF ROTARY INTERNATIONAL

July 21, 12:15 p.m., Luncheon—New York Room, Hotel Statler

President—Charles L. Naylor, D.O., Ravenna, Ohio
Secretary-Treasurer—Russell Peterson, D.O., Phoenix, Ariz.

OSTEOPATHIC WAR VETERANS ASSOCIATION

July 19, 12:30 p.m., Luncheon and Meeting—Rendezvous Room, Biltmore Hotel

President—J. J. O'Connor, D.O., Toronto, Ontario, Canada (Deceased)
Secretary-Treasurer—True B. Eveleth, D.O., Chicago

OSTEOPATHIC WOMEN'S NATIONAL ASSOCIATION

July 17, 3:00-5:00 p.m., Executive Board Meeting—Cleveland Room, Hotel Statler

7:00 p.m., Banquet—Mission Room, Hotel Statler

July 20, 12:15 p.m., Luncheon—New York Room, Hotel Statler

President—Louise W. Astell, D.O., Champaign, Ill.
Secretary-Treasurer—Arvilla McCall Rose, D.O., Evanston, Ill.

SOCIETY OF DIVISIONAL SECRETARIES

July 15, 9:00 a.m.-5:00 p.m., Meeting—Conference Room No. 4, Biltmore Hotel

July 16, 12:30 p.m.—Luncheon and Meeting with Association of Osteopathic Publications—Conference Room No. 8, Biltmore Hotel

President—P. Ralph Morehouse, D.O., Albion, Mich.
Secretary-Treasurer—Mr. George W. Thomas, Harrisburg, Penna.

Friday, July 15

Morning

9:30 Budgets for Small Societies
David E. Reid, D.O., Lebanon, Ore.
9:45 Discussion
10:00 Delinquent Members and Unpaid Dues
R. M. Packard, D.O., Jonesboro, Ark.
10:15 Discussion
10:30 Retirement and Pension Plans for Divisional Society Employees
Mr. Lloyd Hall, Topeka, Kans.
10:50 Discussion
11:10 Technics of Stimulating the Living Endowment Program
Mr. Lewis F. Chapman, Chicago
11:25 Discussion
11:40 Round Table Discussion
Unfinished business and other topics not covered

Afternoon

2:00 Legislative Problems and Technics
Mr. Paul D. Adams, Jefferson City, Mo.
2:30 Discussion
2:45 Divisional Society Problems as Observed by the Executive Secretary
R. C. McCaughan, D.O., Chicago
3:15 Discussion
3:30 Developing a Manual of Procedure
Mr. Walter Gray, Oklahoma City, Okla.
3:45 Discussion
4:00 Round Table Discussion
1. Should there be a Society of Divisional Society Secretaries?
2. A study of relations between Society of Divisional Society Secretaries and House of Delegates
3. The Divisional Society Secretary of the Future

Saturday, July 16

9:00 Problems in Education at the State Level
Mr. Lawrence Mills, Chicago
9:20 Discussion
9:35 The Program of the Division of P. and P.W.
Mr. D. David Darland, Chicago
9:55 Discussion
10:10 Participation in State Public Health Programs
J. M. Moore, D.O., Trenton, Tenn.
10:25 Discussion
10:40 General Public Relations Technics
Mr. Harve Lamont Smith, Highland Park, Mich.
11:10 Discussion
11:30 Annual Meeting
12:30 Luncheon with the Association of Osteopathic Publications

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STILL, A. T., OSTEOPATHIC FOUNDATION AND RESEARCH INSTITUTE

Date and space to be announced

Chairman—Allan A. Eggleston, D.O., Montreal, Canada
Secretary—R. C. McCaughan, D.O., Chicago

Specialty Colleges

AMERICAN COLLEGE OF NEUROPSYCHIATRISTS

July 15, 16, 17, 9:00 a.m.-5:00 p.m., Lecture Sessions—Conference Room No. 9, Biltmore Hotel

July 15, 12:30 p.m., Luncheon, Conclave of Fellows—Conference Room No. 3, Biltmore Hotel

July 15, 7:00 p.m., Banquet—Conference Room No. 5, Biltmore Hotel

President—Floyd E. Dunn, D.O., Kansas City, Mo.
Secretary-Treasurer—Don C. Littlefield, D.O., Long Beach, Calif.

AMERICAN COLLEGE OF OSTEOPATHIC INTERNISTS

July 15, 10:00 a.m.-5:00 p.m., Board of Directors—Conference Room No. 6, Biltmore Hotel

President—Neil R. Kitchen, D.O., Detroit
Secretary-Treasurer—Glennard E. Lahrson, D.O., Oakland, Calif.

AMERICAN COLLEGE OF OSTEOPATHIC OBSTETRICIANS AND GYNECOLOGISTS

July 15, 9:00 a.m.-5:00 p.m., Board of Trustees Meeting—Conference Room No. 7, Biltmore Hotel

July 16, 9:00 a.m.-12:00 noon, Board of Trustees Meeting—Conference Room No. 7, Biltmore Hotel

President—Jerry O. Carr, D.O., Fort Worth, Tex.
Secretary-Treasurer—Harold K. Morgan, D.O., Denver

AMERICAN COLLEGE OF OSTEOPATHIC PEDIATRICIANS

July 15, 9:00 a.m.-12 noon, Clinic Presentations—Los Angeles County Hospital
1:00-5:00 p.m., Lecture Sessions—Conference Room No. 1, Biltmore Hotel
July 16, 9:00 a.m.-12:00 noon, Business Meeting—Conference Room No. 1, Biltmore Hotel
1:30-4:30 p.m., Lecture Sessions—Conference Room No. 1, Biltmore Hotel
6:00 p.m., Reception sponsored by Borden Company—Suite to be announced
7:30 p.m., Banquet—Music Room, Biltmore Hotel
July 17, 9:00 a.m.-12:00 noon, Business Meeting—Conference Room No. 1, Biltmore Hotel

President—Thomas F. Santucci, D.O., Philadelphia
Secretary-Treasurer—Arnold Melnick, D.O., Philadelphia
Program Chairman—Betsy B. MacCracken, D.O., Hollywood, Calif.

Friday, July 15

Morning

9:00-12:00 Clinical Presentations, Los Angeles County Osteopathic Hospital—Pediatric Staff of Los Angeles County Osteopathic Hospital

Afternoon

2:00 Foot Problems in Children and Their Management
Speaker to be announced
2:30 Pediatric Endocrine Diagnosis
Quintos W. Wilson, D.O., Kansas City, Mo.
3:00 Management of the Diabetic Child
Speaker to be announced
4:15 Anesthesia in Children
Glenn F. Gordon, D.O., Glendale, Calif.

Evening

6:30 Dinner meetings of Committees

Saturday, July 16

Morning

9:00-12:00 Business Meeting

Afternoon

1:30 Management of Tetanus
Alex Lourie, D.O., Los Angeles
2:00 X-ray Diagnosis of Problems of the Newborn
Louise F. Swift, D.O., Burbank, Calif.
3:00 Recognition and Management of Infant Allergy
Norman W. Clein, M.D., Seattle, Wash.
4:00 Psychology of Vandalism
Fleda Brigham, D.O., Kirksville, Mo.

Evening

6:00 Reception sponsored by the Borden Company
7:30 Banquet and Installation of Officers

Sunday, July 17

Morning

9:00-12:00 Business Meeting

AMERICAN COLLEGE OF OSTEOPATHIC SURGEONS

July 16, Executive Committee Meeting—Buffalo Room, Hotel Statler
July 17, Finance Committee and Special Committee Meetings—Buffalo Room, Hotel Statler
July 18, 19, Evaluating Committee Meeting for Register of Training Programs—Buffalo Room, Hotel Statler
July 20, 21, 22, 23, Membership Committee Meeting—Buffalo Room, Hotel Statler

President—J. Willoughby Howe, D.O., Hollywood, Calif.
Secretary-Treasurer—Charles L. Ballinger, D.O., Toledo, Ohio
Executive Secretary—Orel F. Martin, D.O., Coral Gables, Fla.

AMERICAN OSTEOPATHIC COLLEGE OF ANESTHESIOLOGISTS

July 17, 9:00 a.m.-5:00 p.m., Executive Committee Meeting—Conference Room No. 3, Biltmore Hotel
12:30 p.m., Luncheon—Conference Room No. 3, Biltmore Hotel

President—J. Calvin Geddes, D.O., Mount Clemens, Mich.
Secretary-Treasurer—Crawford M. Esterline, D.O., Kirksville, Mo.

Alumni Meetings

CHICAGO COLLEGE OF OSTEOPATHY

July 20, 6:30 p.m., Banquet—Space to be announced

President—A. F. Kull, D.O., South Bend, Ind.
Secretary-Treasurer—Ward E. Perrin, D.O., Chicago

DES MOINES STILL COLLEGE OF OSTEOPATHY AND SURGERY

July 20, 7:00 p.m., Banquet—New York Room, Hotel Statler

President—Jean F. LeRoque, D.O., Des Moines, Iowa
Secretary-Treasurer—Donald E. Sloan, D.O., Des Moines, Iowa

KIRKSVILLE COLLEGE OF OSTEOPATHY AND SURGERY

July 19, 20, 8:00-a.m.-10:00 a.m., House of Delegates—Mission Room, Hotel Statler
July 20, 7:00 p.m., Banquet—Pacific Ballroom, Hotel Statler

President—George Cozma, D.O., Cleveland, Ohio
Secretary—Margaret H. Raffa, D.O., Tampa, Fla.

PHILADELPHIA COLLEGE OF OSTEOPATHY

July 20, 7:00 p.m., Banquet—Space to be announced

President—John E. Devine, D.O., Ocean City, N. J.
Secretary—H. Willard Sterrett, Jr., D.O., Philadelphia

COLLEGE OF OSTEOPATHIC PHYSICIANS AND SURGEONS

July 20, 7:00 p.m., Banquet—Sierra Room, Hotel Statler

President—H. W. Wagenseller, D.O., Beverly Hills, Calif.
Secretary-Treasurer—Pauline L. Harris, D.O., San Marino, Calif.

Fraternities and Sororities

NATIONAL OSTEOPATHIC INTERFRATERNITY COUNCIL

July 19, 12:15 p.m., Luncheon—Cleveland Room, Hotel Statler

President—Donald E. Sloan, D.O., Des Moines, Iowa
Secretary-Treasurer—Floyd E. Dunn, D.O., Kansas City, Mo.

ATLAS CLUB

July 19, 7:00 p.m., Banquet—Conference Room No. 4, Biltmore Hotel

President—Henry J. Ketman, D.O., Des Moines, Iowa
Secretary-Treasurer—Robert R. Magliocco, D.O., Milwaukee

AXIS

July 19, 6:00 p.m., Banquet—Washington Room, Hotel Statler

President—Esther Smoot, D.O., Kansas City, Mo.
Secretary—M. Marguerite Fuller, D.O., Cape Girardeau, Mo.

DELTA OMEGA

July 19, 6:30 p.m., Banquet—Cleveland Room, Hotel Statler

President—Mary Leone McNeff, D.O., Farwell, Tex.
Secretary—Betty H. Slaughter, D.O., Lexington, Mo.

IOTA TAU SIGMA

July 17, 2:00 p.m.; July 18-19, 1:00 p.m., Business Meetings—Conference Room No. 5, Biltmore Hotel
July 19, Iota Tau Sigma Night at the Drunkard Theatre

President—Donald E. Sloan, D.O., Des Moines, Iowa
Secretary—A. Leon Sikkenga, D.O., Orlando, Fla.

LAMBDA OMICRON GAMMA

July 21, 12:00 p.m., Luncheon—Renaissance Room, Biltmore Hotel

President—Sidney Slotkin, D.O., Collingswood, N. J.
Secretary—Aaron A. Feinstein, D.O., Philadelphia

PHI SIGMA GAMMA

July 18, 12:15 p.m., Luncheon for Executive Committee—Cleveland Room, Hotel Statler

July 19, 6:30 p.m., Reception—Mission Room, Hotel Statler

7:30 p.m., Banquet—Mission Room, Hotel Statler

President—Jean F. LeRoque, D.O., Des Moines, Iowa
Secretary-Treasurer—Floyd E. Dunn, D.O., Kansas City, Mo.

PSI SIGMA ALPHA

July 20, 8:00 a.m., Breakfast—Washington Room, Hotel Statler

President—Mervin E. Meck, D.O., Oak Hill, W. Va.
Executive Secretary-Treasurer—John W. Hayes, D.O., East Liverpool, Ohio

SIGMA SIGMA PHI

July 17, 5:30 p.m., Business Meeting—Washington Room, Hotel Statler

6:30 p.m., Reception—Cleveland Room, Hotel Statler

President—S. V. Robuck, D.O., Chicago
Secretary-Treasurer—P. R. Koogler, D.O., Kirksville, Mo.

THETA PSI

July 19, 7:00 p.m., Banquet—New York Room, Hotel Statler

President—Kirk L. Hilliard, D.O., Pleasantville, N. J.
Secretary-Treasurer—E. P. Carlton, D.O., Ft. Worth, Tex.

Service Clubs

QUOTA CLUB

July 20, 7:00 a.m., Breakfast—Cleveland Room, Hotel Statler

Program Chairman—Georgia B. Hager, D.O., Los Angeles

LOCAL CONVENTION COMMITTEE

General Chairman—Robert M. Loveland, D.O.
Honorary Chairman—Louisa Burns, D.O.
Secretary—Betsy B. MacCracken, D.O.
Facilities—John V. Fiore, D.O.
Entertainment—Donald M. Donisthorpe, D.O.
Entertainment for Women—Mrs. Elmer S. Clark
Clinics—Delmar J. Daniel, D.O.
Information—Basil Harris, D.O.
Transportation—Arthur E. Miller, D.O.
Golf—Robert C. Ruenitz, D.O.
Public Relations—J. Gordon Hatfield, D.O.
Assistants to the Chairman:
J. Claver Cargill, D.O.
George Z. DuPont, D.O.
Edwin H. Riedell, D.O.
Program Chairman, 1955—W. Donald Baker, D.O.
Program Chairman, 1956—Myron C. Beal, D.O.
Program Chairman, 1957—Neil R. Kitchen, D.O.

ANNUAL CONVENTION REGISTRATION RULES

The following are the rules for registration for the Fifty-Ninth Annual Convention of the American Osteopathic Association, Los Angeles, July 5-24, 1955.

Those who may register are: members of the Association, their children, and their adult guests who are not osteopathic physicians; osteopathic students; commercial and scientific exhibitors; nonmembers of the Association eligible for membership; nonmembers who are ineligible for membership, but who show written evidence of membership in a divisional society; and employees of the Association and of the Los Angeles Convention Committee.

However, not all classes of registrants may attend all sessions of the convention. While all registrants may attend the general sessions, adult and juvenile guests and commercial exhibitors may not attend the special group instruction meetings. All other privileges dependent upon the registration fee will be granted to these guests.

The registration fees will be $18 (including tax) for members attending the Teaching Sessions only, July 18-22, and for adult guests. Special qualifications are required for those registering for the Instruction Courses, July 5-16, and an additional fee will be charged, the amount depending on the course selected.

Osteopathic physicians not eligible for membership in the Association may register for the convention Teaching Sessions (July 18-22, inclusive), but only upon the presentation of official, written evidence of current membership in a divisional society of the Association. These doctors must pay a fee of $25, in addition to the $18 convention registration fee.

Doctors of osteopathy who are not members of the Association, but appear to be eligible for membership, must pay a fee of $75, in addition to the $18 convention registration fee.

These doctors may apply for membership at the registration desk and their $75 fee will be applied as their annual dues. All such applications will be put through the regular channels. If the applicant is not acceptable, $50 of the $75 will be returned and the remainder retained as the registration fee charged ineligible nonmembers.

Divisional societies include state and provincial societies and the British and Australian Osteopathic Associations. Membership in a local, city, county, or district society is not sufficient for registration eligibility.

R. C. McCaughan, D.O.
Executive Secretary

RULES CONCERNING A.O.A. CONVENTION INVITATIONS

In relation to the formal invitation to this Association to hold an annual convention in a particular place, the Constitution of the American Osteopathic Association was amended at the 1951 convention to provide that "the House may take action covering not more than five succeeding conventions." Therefore, it will be possible, but not mandatory, for the House of Delegates at the 1955 convention in Los Angeles to select the convention city for the 1958, 1959, and 1960 conventions.

Formal invitations must be received not less than 60 days before the convention and in such invitation the inviting city is required to give detailed description of physical facilities available and of a local organization now set up or to be set up. The descriptive information is an essential part of the convention invitation, and failure to provide it will bar consideration of the invitation by the Convention City Committee and by the House of Delegates. The Convention City Committee is composed of E. H. McKenna, Chairman, S. H. Leibov, Lawrence C. Boatman, R. C. McCaughan, and C. N. Clark.

Following are the formal rules involved:

"The invitations shall be received not less than sixty days before the time of selection of convention city so that plenty of time may be offered for investigation. Inviting cities shall in

their invitation give detailed description of physical facilities and of their local organization. These descriptions shall be exact and failure to provide this information to the Committee shall bar consideration of the invitation by the Committee and the House of Delegates.

"The Committee shall report its findings, recommendations, and reasons therefor to the Board and the House of Delegates early in the annual sessions each year so that adequate time may be had for discussion before a decision is arrived at on location by the House. The House shall give serious consideration to the recommendation of the Committee. The Committee may recommend one city or more than one if two alternatives seem equally acceptable. In recommending a convention city the Committee shall consider the following factors listed in the order of their importance:

(a) Physical facilities for general and sectional and affiliated groups' programs, and for housing attendants.
(b) Location of city for drawing largest attendance.
(c) Clinical facilities (legal status of profession).
(d) Location of city as drawing card for exhibitors.
(e) Physical facilities as a drawing card for exhibitors.
(f) Expense to the Association in putting on convention.
(g) Intraprofessional good will promotion, here considering location and success of previous conventions.
(h) Public relations of the profession.
(i) Ability of the local group to provide the necessary local committee.
(j) Available entertainment for members' families.

"Divisional societies or associations extending convention city invitations shall be requested to give detailed description of physical facilities of their city and their local organization. All such communications shall be presented to the Convention City Committee not less than sixty days before the time of selection of the convention city."

R. C. McCaughan, D.O.
Executive Secretary

Proposed Amendments to the Constitution and Bylaws and Code of Ethics of the American Osteopathic Association

R. C. McCAUGHAN, D.O.
Executive Secretary

CONSTITUTION

(The following proposed amendment is published at the request of the Executive Secretary in order to correct a grammatical error. This amendment will be read to the 1955 House of Delegates but cannot be acted upon until 1956.)

Article X—Amendments

Amend Article X by deleting in the last line of the article the words, "it is," and substituting therefor the words, "they are." The article would then read: "This Constitution may be amended by the House at any annual meeting, by a two-thirds vote of the accredited voting Delegates at such meeting, provided that such amendments shall have been presented to the House and filed with the Secretary at a previous annual meeting, and that the Secretary shall have them printed in THE JOURNAL not less than two months nor more than four months previous to the meeting at which they are to be acted upon."

BYLAWS

(The following proposed amendment is published at the direction of the Board of Trustees on recommendation of the Bureau of Hospitals.)

Article II—Membership

Amend Section 1 by deleting in the first sentence the words, ", licensed to practice in the state from which he applies," and inserting after the word "society" the words "in the area." The sentence would then read: "An applicant for regular membership in this Association shall be a graduate of a recognized college of osteopathy and shall make application upon the prescribed form with the endorsement of the secretary of the divisional society in the area in which the applicant resides."

(The following amendment is published at the direction of the Board of Trustees in order to clarify the establishment and amendment of the Code of Ethics.)

Article III—Code of Ethics

Amend the Bylaws by adding a new Article III as follows:

Section 1. The House of Delegates shall establish a Code of Ethics for the information and guidance of the members. Members of the Association shall comply, in their daily conduct, with the provisions of the Code of Ethics. The Code shall cover duties of physicians to patients, duties of physicians to the profession at large and to the other members thereof, and responsibilities of physicians to the public.

The House of Delegates shall not adopt any provision of the Code of Ethics in conflict with the Constitution or Bylaws of the Association.

Section 2. The Code of Ethics may be amended at any annual meeting of the House by two-thirds vote of the accredited voting delegates provided a copy of said amendment be deposited with the Secretary at least 60 days before the annual meeting at which the said amendment is to be voted upon. It shall be the duty of the Secretary to have the proposed amendment published in THE JOURNAL of the Association not later than one month before the annual meeting at which the amendment is scheduled for consideration.

Article III to Article XI

Further amend the Bylaws by changing the present Article III to Article IV, Article IV to Article V, Article V to Article VI, Article VI to Article VII, Article VII to Article VIII, Article VIII to Article IX, Article IX to Article X, and Article X to Article XI.

(The following proposed amendment is published at the direction of the Board of Trustees. It would delete the Committee on Professional Visual Education.)

Article IX—Departments, Bureaus, Committees and Sections

Amend Section 1 by deleting, in the first sentence, the words, ", Research, and the Committee on Professional Visual Education," and by substituting therefor the words ", and Research." The sentence would then read: "The Department of Professional Affairs shall include the Bureaus of Professional Education and Colleges, Hospitals, Conventions, Professional Development, and Research."

(The following proposed amendment is published to place in the Bylaws a bureau established at the direction of the Board of Trustees.)

Article IX—Departments, Bureaus, Committees and Sections

Amend Section 1, by inserting in the first sentence of the Section, following the word "Research", the words "the Bureau for Evaluation of Therapeutic Modalities,".

CODE OF ETHICS

(The following proposed amendment is published at the direction of the Board of Trustees with the recommendation that it be approved. It would relieve the Committee on Ethics and Censorship of the formality of approving the distribution of reprints of articles that appear in THE JOURNAL of the Association.)

Chapter II, Article I—Duties for the Support of Professional Character

Amend Section 9, by deleting in line 9 thereof, the words, "and the Committee on Ethics and Censorship."

American Osteopathic Association

ADVANCE REGISTRATION BLANK

Annual Convention—Los Angeles
July 5-24, 1955

Dr.....
(Please print name)

.....
(Street address) (City) (Zone) (State)

REGISTRATION FEES

CONVENTION INSTRUCTION COURSES—July 5 to July 16, inclusive: Registration for the Convention Teaching Sessions is required of all who register for any of the Convention Instruction Courses.

	Instruction Course	Teaching Session	TOTAL
Section I—Advanced Surgery	$232.00	$18.00	$250.00
Section II—Regular Surgery	$132.00	$18.00	$150.00
Section III—Internal Medicine and Cardiology	$132.00	$18.00	$150.00
Section IV—General Practice and Peripheral Vascular Disease	$ 57.00	$18.00	$ 75.00
Section V—Anesthesiology	$132.00	$18.00	$150.00

CONVENTION TEACHING SESSIONS, ONLY—July 18 to July 22, inclusive:

Member of American Osteopathic Association	$18.00	$ 18.00

GUESTS OF MEMBERS—July 18 to July 22, inclusive:

Adult Guests (Name)	$18.00	$ 18.00
Juvenile Guests (Name) (Under 18 years)	No Fee	No Fee

APPLICATION FORM

CONVENTION INSTRUCTION COURSES:

Indicate Choice (1st, 2nd, and 3rd)		Fees	
.....	Section I—Advanced Surgery	$250.00	.....
.....	Section II—Regular Surgery	$150.00	.....
.....	Section III—Internal Medicine and Cardiology	$150.00	.....
.....	Section IV—General Practice and Peripheral Vascular Disease	$75.00	.....
.....	Section V—Anesthesiology	$150.00	.....
	CONVENTION TEACHING SESSIONS, ONLY	$18.00	.....
	ADULT GUESTS	$18.00	.....
	JUVENILE GUESTS	No Fee	.....
	Total		$.....

RESUME OF YOUR PROFESSIONAL AND GRADUATE TRAINING

Preprofessional or Academic Training (Where obtained, date): .....

.....

.....

Degrees (Where obtained, date): .....

.....

Member (or Candidate) of specialty society, specialty college, Academy of Applied Osteopathy, American College of General Practitioners in Osteopathic Medicine and Surgery, *et al.* (designate) .....

.....

Certified in .....

Member of additional specialty groups (designate) .....

.....

Additional postgraduate courses completed (designate) .....

.....

This space for A.O.A. Central Office use, only:

Amount Received .....

Date postmarked .....

Date received .....

REGISTRATION RULES—1955

CONVENTION INSTRUCTION COURSES:

Section I—Advanced Surgery: Limited to members of the American College of Osteopathic Surgeons or certified surgeons whose background and experience in general surgical procedures will enable them to readily acquire the specialized technics of cardiovascular surgery.

Section II—Regular Surgery: Limited to members or candidates of the American College of Osteopathic Surgeons or individuals eligible to such.

Section III—Internal Medicine and Cardiology: Limited to osteopathic physicians and surgeons who are members or candidates of the American College of Osteopathic Internists or are eligible to such.

Section IV—General Practice and Peripheral Vascular Disease: This course is designed for all general practitioners in osteopathic medicine and surgery. Any member of the American Osteopathic Association is eligible.

Section V—Anesthesiology: Limited to members of the osteopathic profession who are either certified in Anesthesiology, in training for such certification, or are spending a portion of their time giving anesthetics and are interested in improving their skill and learning new anesthetic technics.

METHOD OF ASSIGNMENT TO CONVENTION INSTRUCTION COURSES:

The Committee on Convention Instruction Courses, shall, insofar as possible, consider each applicant on the basis of: (1) eligibility and (2) the date of mailing of the application.

The Committee on Convention Instruction Courses will be the sole judge of the eligibility of each applicant for enrollment in the course of choice.

CONVENTION TEACHING SESSIONS, ONLY:

Those who may register are: members of the Association, their children, and their adult guests who are not osteopathic physicians; osteopathic students; osteopathic students' wives; commercial and scientific exhibitors; nonmembers of the Association eligible for membership; nonmembers who are ineligible for membership, but who show written evidence of membership in a divisional society; and employees of the Association and of the Los Angeles Convention Committee.

Make Hotel Reservations Now!

59th Annual Convention
American Osteopathic Association
Convention Instruction Courses — July 5-16, 1955
Teaching Sessions — July 18-24, 1955
Los Angeles, California

HOTEL RATES*

HOTELS	Single	Double	Twin Beds	Triple	Suites
STATLER Wilshire and Figueroa	$ 6.50 $12.00	$ 9.00 $14.00	$10.00 $15.50	$ 2.50 extra per person	$19.00 up
BILTMORE 515 S. Olive Street	$ 6.00 $11.50	$ 8.50 $14.00	$10.00 $14.50	$ 2.50 extra per person	$20.00 $30.00
NEW CLARK 426 S. Hill Street	$ 4.50 $ 6.50	$ 6.00 $ 8.00	$ 7.00 $ 9.00	$ 9.00 $12.00	$12.00 $18.00
AMBASSADOR 3400 Wilshire Blvd	$ 9.00 $23.00	$12.00 $26.00	$12.00 $26.00	$ 3.00 extra per person	$30.00 $48.00

NOTE: Single rooms are extremely limited. You will have a much better chance of obtaining accommodations if you request rooms that are to be occupied by two or more persons.

*Subject to change.

Important!

Please read these instructions before filling out application blank on next page.

(1) All reservations must be made directly through the A.O.A. Housing Bureau:

Convention and Visitors Bureau
Los Angeles Chamber of Commerce
1151 South Broadway
Los Angeles 15, California

(2) Do not send reservations to the local convention arrangements committee or to the American Osteopathic Association Central Office.

(3) Classifications of eligible applicants for hotel accommodations: member, officer, trustee, delegate, alternate, scientific or technical exhibitor.

(See hotel reservation blank on next page)

APPLICATION FOR HOTEL ACCOMMODATIONS

American Osteopathic Association Convention

Convention Instruction Courses . . . July 5-15, 1955

Teaching Sessions . . . . . July 18-24, 1955

Los Angeles, California

(See reverse side of page for instructions and hotel rates)

A.O.A. Housing Bureau
Convention and Visitors Bureau
Los Angeles Chamber of Commerce
1151 South Broadway
Los Angeles 15, California

Date of Application.....

Accommodations Desired
(Name of Hotel)

First choice.....
(Print)

Second choice.....

Third choice.....

(If the hotels of your choice are unable to accept your reservation, the A.O.A. Housing Bureau will make as good a reservation as possible elsewhere, providing all hotel rooms have not already been taken.)

Single room.....Room with double bed.....Room with twin beds.....

Date and hour of arrival.....Date and hour of departure.....

Guests' Names

The name of each hotel guest must be listed. Therefore, please include the names of both persons who will occupy each double room requested. Please do not make reservations for anyone without definite agreement with parties involved. The name and address of each person for whom you are requesting reservations and who will occupy the room is:

.....

.....

.....

Individual Requesting Reservations

Name.....

Address.....

City and State.....

Name of firm, if technical exhibitor.....

All reservations must be received prior to June 20, 1955!

The Technical Exhibitors

CLAYTON N. CLARK, D.O.
Manager of Exhibits

The following firms have reserved exhibit space for the Los Angeles Convention. We urge the profession to visit the booths of these companies, not only to show appreciation for their cooperation and support but to obtain highly interesting and very useful information concerning the wide variety of products which will be on display.

HOTEL STATLER, LOS ANGELES—JULY 18-22

Technical Exhibitors	Exhibit Space
ABBOTT LABORATORIES North Chicago, Ill.	25
AIR SHIELDS, INC. Hatboro, Pa.	7
A. S. ALOE CO. OF CALIF. 1150 S. Flower St., Los Angeles 15, Calif.	76
AMERICAN FERMENT CO. 1450 Broadway, New York 18, N. Y.	91
AMERICAN HOSPITAL SUPPLY CO. 2220 Ridge Ave., Evanston, Ill.	22
AMERICAN STERILIZER CO. 1230 Plum St., Erie, Pa.	93
AMES COMPANY, INC. Elkhart, Ind.	95
ARMOUR LABORATORIES Kankakee, Ill.	70
AYERST LABORATORIES 22 E. 40th St., New York 16, N. Y.	41
BAKER LABORATORIES, INC. 4614 Prospect Ave., Cleveland 3, Ohio	58
BASIC ENDOCRINE SALES, INC. 406 Loew State Bldg., Los Angeles 14, Calif.	74
BILHUBER-KNOLL CORP. Crane St., Orange, N. J.	85
THE BIRTCHER CORP. 4371 Valley Bldg., Los Angeles 32, Calif.	14-15
BORCHERDT MALT EXTRACT CO. 217 N. Wolcott Ave., Chicago 12, Ill.	96
THE BORDEN CO. 350 Madison Ave., New York 17, N. Y.	84
BOYLE & CO. 356 S. Mission Rd., Los Angeles 33, Calif.	23
BRISTOL LABORATORIES 630 5th Ave., New York 20, N. Y.	30
BRISTOL-MYERS CO. 630 Fifth Ave., New York 20, N. Y.	40
BROEMMEL PHARMACEUTICALS 1235 Sutter St., San Francisco 9, Calif.	53
A. M. BROOKS CO. 1222 W. Washington Blvd., Los Angeles 7, Calif.	6
BUFFALOW MFG. CO. 915 Volunteer Bldg., Chattanooga 2, Tenn.	75
BURDICK CORP. Milton, Wis.	94
BURTON MFG. CO. 11201 W. Pico Blvd., Los Angeles 64, Calif.	24
CAMERON SURGICAL SPECIALTY CO. 666 W. Division St., Chicago 10, Ill.	18
S. H. CAMP & CO. Jackson, Mich.	64
CARNATION CO. Box 2035 Wilshire-Labrea Sta., Los Angeles 36, Calif.	80
CENTRAL PHARMACAL CO. Seymour, Ind.	48
CHICAGO PHARMACAL CO. 5547 N. Ravenswood Ave., Chicago 40, Ill.	67
COLE CHEMICAL CO., INC. 3721 Laclede Ave., St. Louis 8 Mo.	29
CORECO RESEARCH CORP. 159 W. 25th St., New York 1, N. Y.	19
CORONET PRODUCTS 1815 Telegraph Ave., Oakland 12, Calif.	59
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DARWIN LABORATORIES 7965 Melrose Ave., Los Angeles 46, Calif.	49
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R. A. FISCHER CO. 525 Commercial St., Glendale 3, Calif.	21
C. B. FLEET CO. 921 Commerce St., Lynchburg, Va.	50
E. FOUGERA & CO., INC. 75 Varick St., New York 13, N. Y.	46
GENERAL ELECTRIC CO. 1225 North Vermont St., Los Angeles 29, Calif.	94-A
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GRAVITATOR Mulberry & Cathedral Sts., Baltimore 1, Md.	47
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HILL LABORATORIES Lincoln Highway, Malvern 2, Pa.	44-A
HOLLAND RANTOS CO., INC. 145 Hudson St., New York 13, N. Y.	9
JACUZZI BROS., INC. 1440 San Pablo Ave., Berkeley, Calif.	39
JOHNSON & JOHNSON 501 George St., New Brunswick, N. J.	73
MEAD JOHNSON & CO. Evansville 21, Ind	4
LEDERLE LABORATORIES Pearl River, N. Y.	33
LIEBEL-FLARSHEIM CO. 111 E. Amity Road, Cincinnati 15, Ohio	92
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McMANIS TABLE CO. Kirksville, Mo.	63
MEDCO PRODUCTS CO. 3607 E. Admiral Pl., Tulsa 12, Okla.	34
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SANBORN CO. Cambridge 39, Mass.	60
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JULIUS SCHMID, INC. 423 W. 55th St., New York 19, N. Y.	28
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STUART CO. 234 E. Colorado St., Pasadena 1, Calif.	8
SUNKIST GROWERS 707 W. Fifth St., Los Angeles 54, Calif.	87
TESTAGAR CO. 1354 W. La Fayette, Detroit 26, Mich.	12
THURSTON LABORATORIES, INC. 2457 Fletcher Dr., Los Angeles 39, Calif.	2
THE TORRANCE CO. Saniwax Bldg., Kalamazoo, Mich.	57
S. J. TUTAG & CO. 19180 Mt. Elliott, Detroit 34, Mich.	62
U. S. VITAMIN CORP. 250 East 43rd St., New York 17, N. Y.	73-A
VITAMINERALS, INC. 1816 Flower St., Glendale 1, Calif.	61
WAITE PHARMACAL CO., INC. 5443 N. Broadway, Chicago 40, Ill.	35
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WHITE LABORATORIES Kenilworth, N. J.	17
WINTHROP STEARNS, INC. 1450 Broadway, New York 18, N. Y.	72
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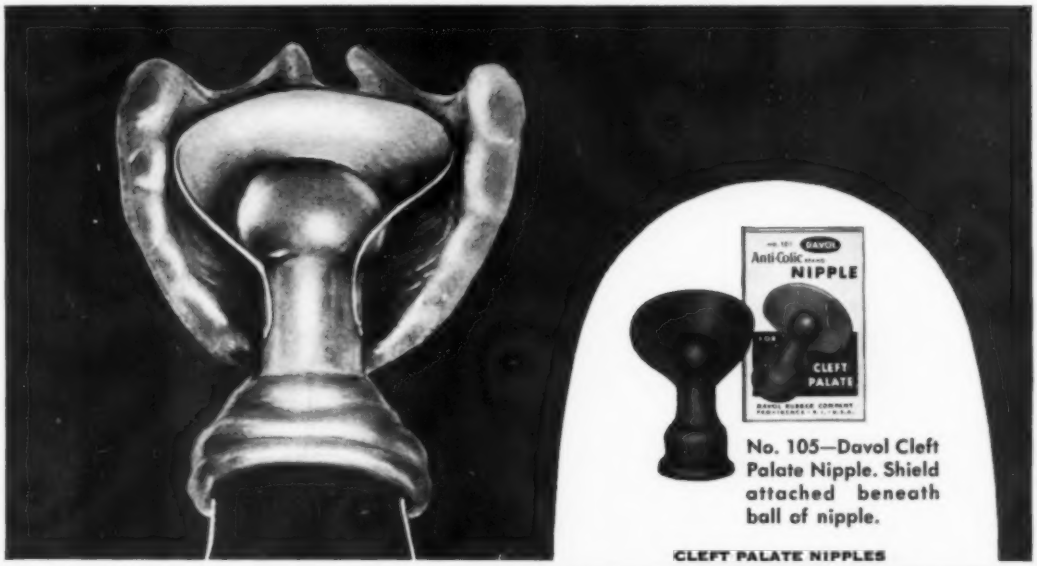

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up against this stubborn trio?

nervous tension
associated pain
smooth muscle spasm

IMPLEMENT YOUR TREATMENT WITH EFFECTIVE

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BY THE CONCERTED ACTION of the natural alkaloids of belladonna and the mild, antispasmodic effect of CLORTRAN, BUTADONNA provides at once spasmolysis, sedation and a local anesthetic effect on the gastrointestinal mucosa. The belladonna alkaloids, asserts W. T. Salter, "have withstood the 'acid test' of medical experience over the centuries." Combined with these in management of hypermotility and spasm, an exceptionally rational choice is CLORTRAN — non-barbiturate sedative without side effects, which allays excitement centrally and eases pain peripherally.*

**Textbook of Pharmacology, (Saunders) 1952.*

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Clortran (Chlorobutanol)	0.25 gm. (3-3/4 gr.)
Hyoscyamine Sulfate	0.1 mg.
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Total Alkaloidal salts	0.127 mg. (1/500 gr.)

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NEW SPASMOLYTIC-SEDATIVE

DOSAGE: One capsule three times a day, half hour before meals. As a night time spasmolytic-sedative, two or more capsules at bedtime.
SUPPLIED: Bottles of 100.

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Sedative-Hypnotic-Antinauseant

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- residual symptoms of the barbiturates,
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*Steigmann, F., and Goldberg, E.: Ambulatory Continuous Drip Method in the Treatment of Peptic Ulcer, Am. J. Digest. Dis. 22:67 (Mar.) 1955.

†Mg trisilicate 3.5 gr.; Ca carbonate 2.0 gr.; Mg oxide 2.0 gr.; Mg carbonate 0.5 gr.

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Effective and physiologically compatible nasal vasoconstrictor.

Solution 0.05% in 1-fluidounce bottles with droppers, and in pints.

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Nebulizer, 15 ml. Privine hydrochloride Solution 0.05%.

Also available: Privine Ophthalmic Solution, 0.1%, in 15-ml. bottles with droppers.

Privine® hydrochloride (naphazoline hydrochloride CIBA)

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PENTOXYLON®

...and in a matter of weeks

angina loses its terror

- Reduces nitroglycerin need
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- Increases exercise tolerance
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Each tablet of Pentoxylon combines the tranquilizing, stress-relieving, bradycrotic effects of 1 mg. Rauwiloid and the prolonged coronary vasodilating effect of 10 mg. pentaerythritol tetranitrate (PETN). The combination provides a new completeness of treatment previously unavailable to patients with angina. The contained Rauwiloid serves to overcome tachycardia—to permit better coronary filling without increasing cardiac work. This effect, together with the lasting coronary relaxation afforded by PETN, combine to reduce nitroglycerin needs, increase exercise tolerance, reduce anxiety, allay apprehension and produce objective improvement demonstrable by ECG.

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Descriptive literature on request

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F. M. R. Walshe, M.D., F.R.C.P.

8th ed. 372 pp. 91 figs. $7.00

The eight editions of DISEASES OF THE NERVOUS SYSTEM since its first publication in 1940 prove the book's popularity. In the eighth edition new material reflecting new knowledge and the trend of opinion in clinical neurology has been included. Deletion of older material has prevented increase in length. Critical consideration has been given to new theories to stimulate the critical thinking of the reader, one of the aims of the book.

Local Analgesia: Brachial Plexus

R. R. Macintosh, F.R.C.S., and William W. Mushin, M.R.C.S.

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Pictures give the reader of this small manual an eyewitness lesson in the use of local anesthesia. Thirty-nine illustrations (15 in color) and a clearly written text provide a concise, valuable approach to the subject.

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Although the symptomatology and morbid anatomy of multiple sclerosis are well known, the causation is still unknown, and treatment is still relatively ineffective. The authors discuss in this book what recent advances there have been in the field. Their object is to widen the general conception of the disease, to provide an account of early symptomatology and treatment and to review the pathology and the relationship of multiple sclerosis to other demyelinating diseases, and thus to encourage further research in the field.

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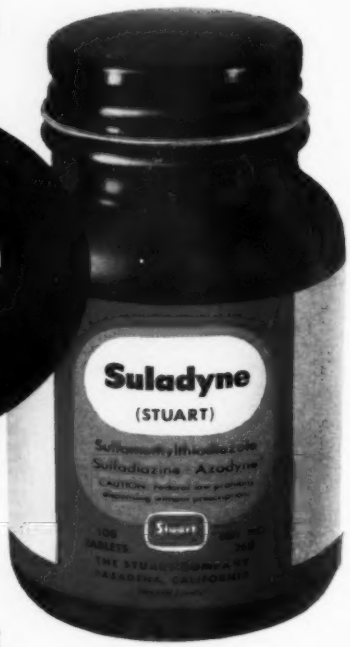

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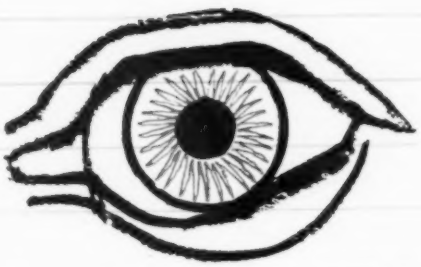
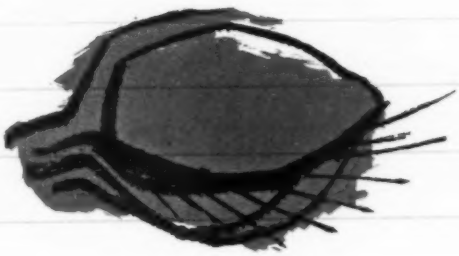


for daytime tension



and nervous insomnia

NOT A BARBITURATE

for daytime tension and nervous insomnia

You can now prescribe Noludar 'Roche' -- a new, mild sedative-hypnotic which is not a barbiturate. Tests in over 3000 patients have confirmed the clinical value of Noludar T.M. (3,3-diethyl-5-methyl-2,4-piperidinedione).

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Fewer recurrences in PSORIASIS

I=76% E=38% R=19% with RIASOL

The most discouraging feature of psoriasis is recurrence. Ormsby and Montgomery* write: "The disease often recurs, and may do so repeatedly for the greater part of a lifetime."

Clinical investigation shows that in psoriasis treated with RIASOL recurrence is exceptional. In a series of resistant psoriatics, classified as therapeutic failures to other drugs, there was improvement with RIASOL in 76% of cases, eradication of the lesions in 38%, and recurrence in only 19%. Such recurrences responded readily to further treatment with RIASOL.

The explanation for these unusual results is to be found in the deep action of RIASOL in the prickle-cell layer of the stratum mucosum, from which the cutaneous lesions of psoriasis originate.

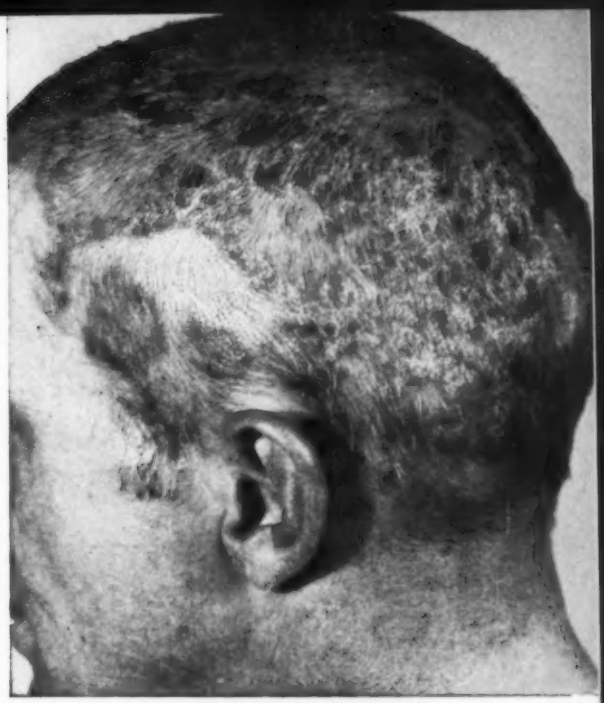
RIASOL contains 0.45% mercury chemically combined with soaps, 0.5% phenol and 0.75% cresol in a washable, non-staining, odorless vehicle.

Apply daily after a mild soap bath and thorough drying. A thin, invisible, economical film suffices. No bandages required. After one week, adjust to patient's progress.

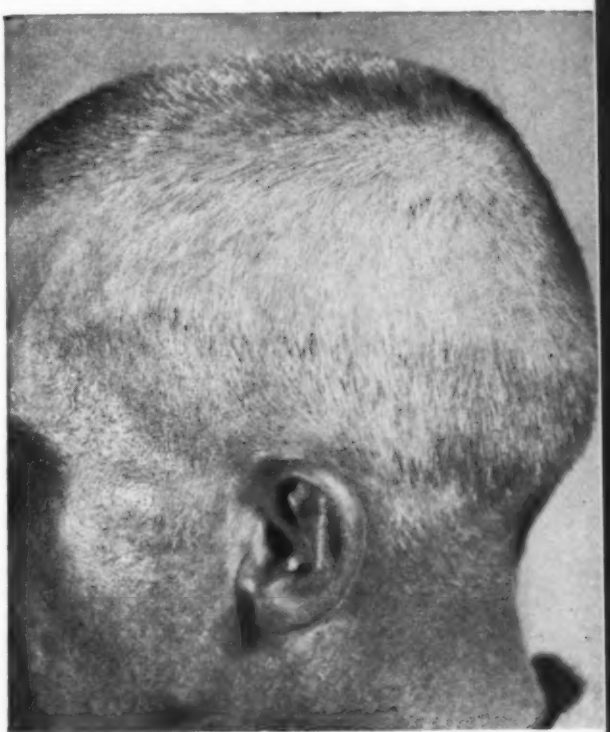
RIASOL is supplied in 4 and 8 fld. oz. bottles at pharmacies or direct.

*Ormsby, O. S. & Montgomery, H., *Diseases of the Skin*, 6th ed., 1943, p. 291.

MAIL COUPON TODAY—
TEST RIASOL YOURSELF



Before Use of Riasol



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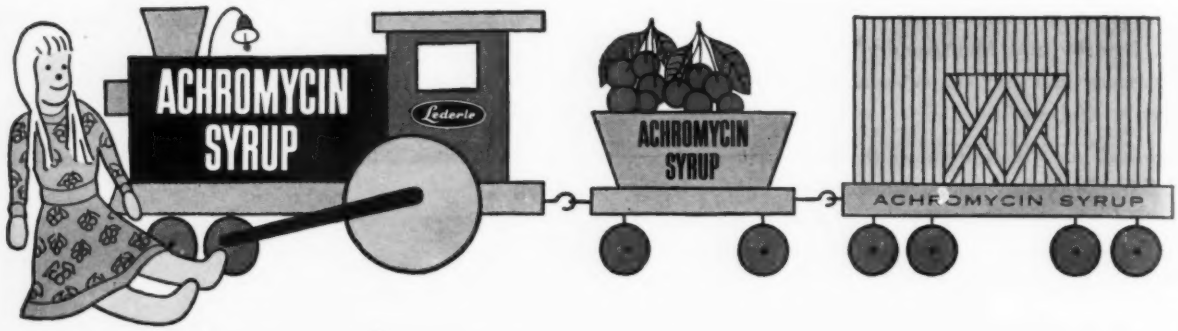
READY TO USE! IN 2 OZ. BOTTLES! AQUEOUS—NO OIL.
NO AFTERTASTE! MISCIBLE WITH WATER, MILK, SODA!



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Oral Suspension (Cherry Flavor): 250 mg. per teaspoonful (5 cc.)

SPERSOIDS* Dispersible Powder (Chocolate Flavor): 50 mg. per rounded teaspoonful (3 Gm.)

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Conventions and Meetings

Announcements

American Osteopathic Association, Fifty-Ninth Annual Convention, College of Osteopathic Physicians and Surgeons and Biltmore and Statler Hotels, Los Angeles; Convention Instruction Courses, July 5-16; Convention Teaching Sessions, July 18-22. Program Chairman, W. Donald Baker, 3450 W. 43rd St., Los Angeles 8.

Academy of Applied Osteopathy, annual meeting, Statler Hotel, Los Angeles, July 22-23. Program Cochairmen, Margaret H. Raffa, 5009 Central Ave., Tampa 3, Fla., and Paul K. Theobald, 1419 Broadway, Oakland 12, Calif. Secretary, Margaret W. Barnes, Box 1345, Carmel, Calif.

American College of Osteopathic Internists, annual meeting, Sheraton-Cadillac Hotel, Detroit, October 20-22. Program Chairman, Ward E. Perrin, Chicago Osteopathic Hospital, 5250 S. Ellis Ave., Chicago 15. Secretary, Glennard E. Lahrson, 460 Staten Ave., Oakland 10, Calif.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Neil House Hotel, Columbus, Ohio, February 6-9. Program Chairman, Richard E. Eby, 1247 N. Park Ave., Pomona, Calif. Secretary, Harold K. Morgan, 3268 W. 32nd Ave., Denver 11.

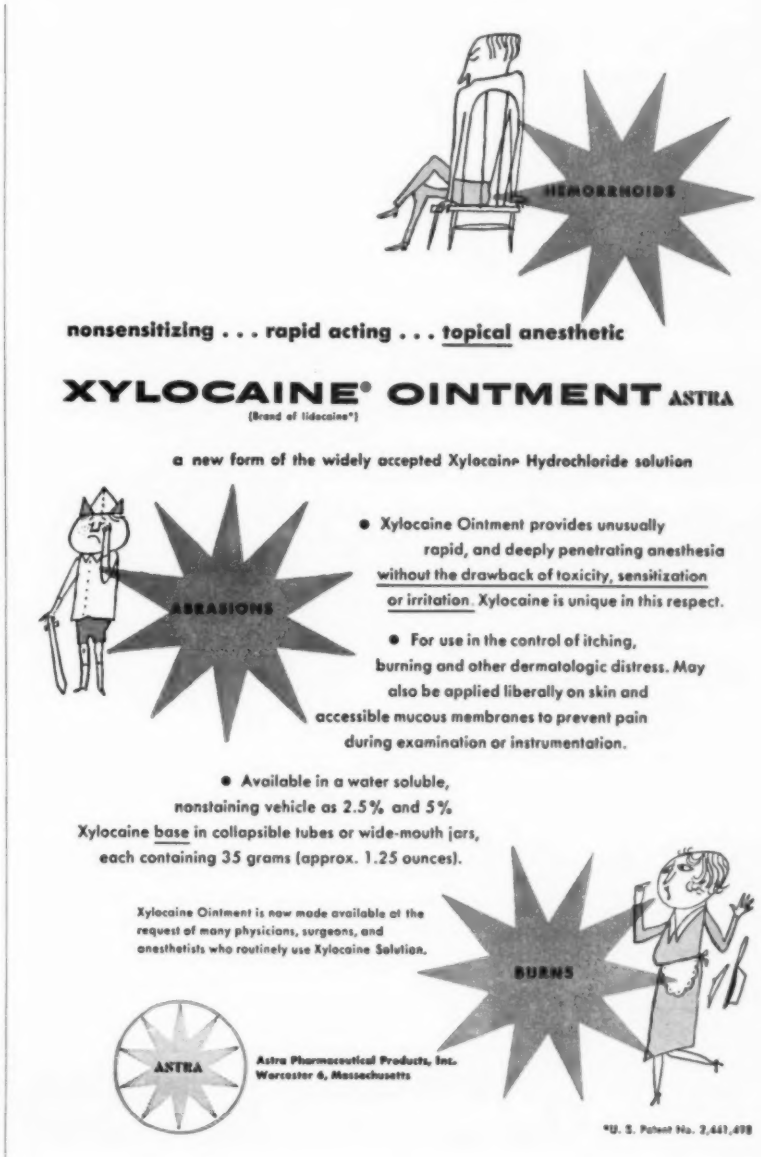
American College of Osteopathic Surgeons, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Program Chairman, Arthur M. Flack, 135 S. 17th St., Philadelphia 3. Executive Secretary, Orel F. Martin, Box 474, Coral Gables 34, Fla.

American Osteopathic Academy of Orthopedics, annual meeting, Hotel Statler, Washington, D. C., October 29-November 2. Program Chairman, Karl P. B. Madsen, 460 Staten Ave., Oakland 10, Calif. Secretary, J. Paul Leonard, 2673 W. Grand Blvd., Detroit 8.

American Osteopathic Academy of Physical Medicine and Rehabilitation, annual meeting, Los Angeles, July 23. Program Chairman, W. Irvin Harner, 302 W. Hadley St., Whittier, Calif. Secretary, John A. Schuck, 1739 Griffin Ave., Los Angeles 31.

American Osteopathic College of Anesthesiologists, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Program Chairman, Harry J. Petri, Jr., 5 Deering St., Portland 3, Maine. Secretary, Crawford M. Esterline, Box 155, Kirksville, Mo.

American Osteopathic College of Radiology, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Program Chairman, A. G. Reed, 212 Pythian Bldg., Tulsa 3, Okla. Secretary, F. A. Turfler, Jr., South Bend Osteopathic Hospital, 118 S. William St., South Bend 2, Indiana.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a new form of the widely accepted Xylocaine Hydrochloride solution
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• For use in the control of itching, burning and other dermatologic distress. May also be applied liberally on skin and accessible mucous membranes to prevent pain during examination or instrumentation.
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Worcester 6, Massachusetts
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American Osteopathic Hospital Association, annual meeting, Hotel Statler, Washington, D. C., October 30-November 2. Program Chairman, Mrs. Alixe P. Nuzum, Des Moines General Hospital, Des Moines, Iowa. Executive Secretary, Mr. R. P. Chapman, 1013 Kahl Bldg., 326 W. Third St., Davenport, Iowa.

Arkansas, annual meeting, Albert Pike Hotel, Little Rock, May 12-13. Program Chairman, L. E. O'Keefe, First State Bank Bldg., Springdale. Secretary, R. M. Packard, 409 Citizens Bank Bldg., Jonesboro.

California, annual meeting, Awahnee Hotel, Yosemite National Park, May 19-22. Program Chairman, Arvel E. Angell, 125 El Tejon Ave., Oildale. Executive Secretary, Mr. Thomas C. Schumacher, 1298 Wilshire Blvd., Los Angeles.

Canadian Osteopathic Association, annual meeting, Mount Royal Hotel, Montreal, October 27-29. Program Chairman, A. E. Wilkinson, 616 Medical Arts Bldg., Montreal 25. Secretary, Miss Joyce C. Currie, 609 Medical Arts Bldg., Montreal 25.

Hawaii, annual meeting, Reef Hotel, Honolulu, July 25-27. Program Chairman, C. W. Wyman, 417 National Bldg., Honolulu 13. Secretary, Joseph W. Stella, 38 Young Hotel Bldg., Honolulu 13.

Indiana, annual meeting, French Lick Springs Hotel, French Lick Springs,

Pediatric—Infant
Reverse Trendelenburg
Varicose Veins
Pediatric—Child
Lateral (Sims)
GYN
Trendelenburg
GU
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Proctologic
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The supreme flexibility of the Ritter Multi-level Table meets every positioning requirement . . . regardless of patient condition, size or age. This motor-elevated table makes your treatment hours easier, you see more patients with less effort. Your Ritter Dealer representative is qualified to give you a complete demonstration of how the Ritter Universal Table can be of greatest value in your practice. Call him now, or write the Ritter Company, Inc., Ritter Park, Rochester 3, N. Y.
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May 15-17. Program Chairman, Paul van B. Allen, 1500 N. Delaware St., Indianapolis 2. Secretary, Arabelle B. Wolf, 809-13 Odd Fellows Bldg., Indianapolis 4.

Iowa, annual meeting, Savery Hotel, Des Moines, May 23-24. Program Chairman, Clive R. Ayers, Grant. Secretary, Mr. Dwight S. James, 200 Walnut Bldg., Des Moines 9.

Kentucky, annual meeting, Brown Hotel, Louisville, October 12-13. Program Chairman, Henry P. Lawrence, Hawesville. Secretary, Harold D. Benteen, 2048 Winchester Ave., Ashland.

Maine, annual meeting, Samoset Hotel, Rockland, June 16-18. Program Chairman, Michael A. Longo, 142 Pine St., Bangor. Midyear meeting, Eastland Hotel, Portland, December 2-3. Secretary, Roswell P. Bates, 72 Main St., Orono.

Massachusetts, annual meeting, Hotel Somerset, Boston, January 21-22. Program Chairman, Everett L. Pierce, Pleasant St., East Dennis. Executive Secretary, Mrs. Gladys M. Stockdale, 524 California St., Newtonville.

Michigan, annual meeting, Civic Auditorium, Grand Rapids, October 3-5. Program Chairman, Harold W. Laidlaw, McCoy-Laidlaw Clinic, Bad Axe. Executive Secretary, Mr. Harve Lamont Smith, 81 Glendale, Highland Park 3.

Missouri, annual meeting, Continental Hotel and Municipal Auditorium, Kansas City, September 27-29. Program Chairman, J. Edward Sommers, 6504A Delmar Blvd., St. Louis 5. Executive Secretary, Mr. Paul D. Adams, 325 E. McCarty St., Jefferson City.

New Mexico, annual meeting, Alvarado Hotel, Albuquerque, May 19-21. Program Chairman, R. K. Widney, Widney Clinic, 1020 Central Ave., S. W., Albuquerque. Secretary, Robert E. Smith, 205 N. First St., Lovington.

New York, annual meeting, Hotel Statler, New York City, October 14-15. Program Chairman, Walter Streicker, 7 Plaza St., Brooklyn 17. Secretary, Robert E. Cole, 417 S. Main St., Geneva.

North Carolina, annual meeting, Battery Park Hotel, Asheville, October 20-22. Secretary and Program Chairman, S. Dales Foster, 710 Public Service Bldg., Asheville.

Northwest Osteopathic Convention, annual meeting, Hotel Multnomah, Portland, Ore., June 13-16. Program Chairman, Russell F. Kenaga, Sr., 2265 W. Burnside St., Portland 5.

Oklahoma, annual meeting, Hotel Tulsa, Tulsa, November 8-10. Program Chairman, Carl R. Samuels, S. Adair St., Box 44, Pryor. Executive Secretary, Mr. Walter L. Gray, 210-12 Braniff Bldg., Oklahoma City.

Oregon: See Northwest Osteopathic Convention.

Osteopathic College of Ophthalmology and Otorhinolaryngology, annual meeting, Continental Hotel, Kansas City, Mo., July 25-27. Program Chairman, H. Mahlon Gehman, 1818 Pine St., Philadelphia 3. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus 15, Ohio.

Pennsylvania, annual meeting, Bellevue-Stratford Hotel, Philadelphia, September 30-October 2. Program Chairman, George S. Esayian, 113 N. Ambler St., Quakertown. Executive Secretary, Mr. George W. Thomas, 1941 Market St., Harrisburg.

South Dakota, annual meeting, Sylvan Lake Hotel, Black Hills, June 5-7. Program Chairman, Laurence S. Betts, Box 205, Huron. Secretary, E. W. Hewlett, 417 W. 27th St., Sioux Falls.

Texas, annual meeting, Rice Hotel, Houston, May 12-14. Program Chairman, J. Natcher Stewart, Stevens Park Osteopathic Hospital, 1141 N. Hampton Road, Dallas 11. Executive Secretary, Phil R. Russell, 512 Bailey St., Fort Worth 7.

Vermont, annual meeting, Long Trail Lodge, Mendon Mt., Rutland, September 28-29. Program Chairman, Hermon K. Sherburne, Jr., Mead Bldg., Rutland. Clerk, Howard I. Slocum, Battell Block, Middlebury.

Virginia, annual meeting, The Lodge, Williamsburg, May 27-28. Program Chairman, Vincent H. Ober, 407-11 Bankers Trust Bldg., Norfolk 10. Acting Secretary, Harold A. Blood, 228 N. Columbus St., Alexandria.

Washington, midyear meeting, Washington Athletic Club, Seattle, December 3-4. Program Chairman, Erskine H. Burton, 710 Jones Bldg., Tacoma 2. Secretary, Eugene E. LaCroix, 1747 S. Sheridan Ave., Tacoma 5.

See also Northwest Osteopathic Convention.

West Virginia, annual meeting, McClure Hotel, Wheeling, June 5-7. Program Chairman, Joseph B. C. Bartram, Gilmer Clinic, Post Office Bldg., Glenville. Secretary, Guy E. Morris, 542 Empire Bank Bldg., Clarksburg.

State and National Boards

ALABAMA

Examinations June 21-23 at the State Capitol, Montgomery Applications must be filed by June 20. Address D. G. Gill, M.D., secretary, Board of Medical Examiners, State Office Bldg., Montgomery 4.

ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

Basic science examinations June 21 at the University of Arizona, Tucson. Applications must be filed 2 weeks prior to the examination. Address Herbert D. Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

COLORADO

Professional examinations June 14-15 at the Y.W.C.A. Auditorium in Denver. Applications must be filed by May 13. Address Mrs. Beulah H. Hudgens, executive secretary, Board of Medical Examiners, 831 Republic Bldg., Denver 2.

CONNECTICUT

Basic science examinations June 11. Applications must be filed 2 weeks prior to the examination. Address Miss M. G. Reynolds, executive assistant, Board of Healing Arts, 52 Whitney Ave., New Haven.

DELAWARE

Examinations July 12-14. Address Joseph S. McDaniel, M.D., secretary, Board of Medical Examiners, 229 S. State St., Dover.

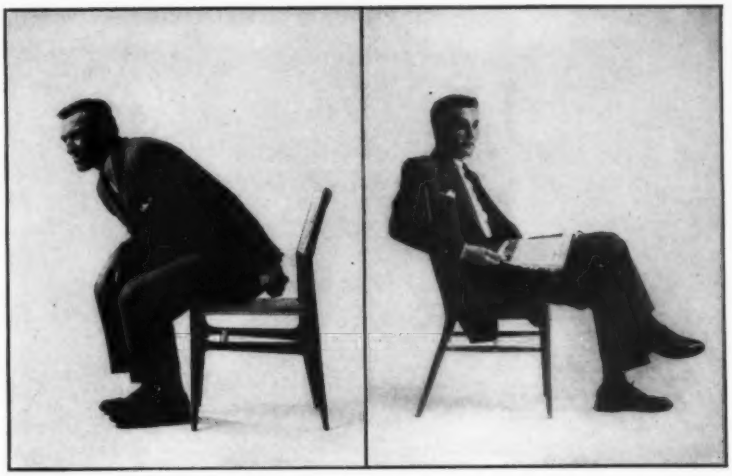
FLORIDA

Professional examinations June 11-12 at the Seminole Hotel, Jacksonville. Applications must be filed prior to May 28. Address R. Philip Coker, D.O., secretary, Board of Osteopathic Medical Examiners, Box 66, Panama City.

GEORGIA

Examinations July 5 at Atlanta. Address Robert K. Glass, D.O., secretary, Board of Osteopathic Examiners, 834-5 Forsyth Bldg., Atlanta 3.

Of two patients with pruritus ani . . .



one is at his wit's end—his lesion excoriated by constant scratching, peace of mind gone, and sleep, too.

The other is symptomatically controlled.

Calmitol makes the difference

Nonsensitizing and free of the dangers of "rebound dermatosis," Calmitol is "preferred"[1] by physicians for its safe and prolonged antipruritic action.

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1½ oz. tubes and 1 lb. jars

1. Lubowe, I. I.: New York State J. Med. 50:1743, 1950.

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IDAHO

Examinations June 9 at Boise. Address D. W. Hughes, D.O., secretary, Board of Osteopathic Examiners, 203 Sun Bldg., Boise.

ILLINOIS

Examinations in June in Chicago. Address Mr. Frederic B. Selcke, Superintendent of Registration, Department of Registration and Education, State House, Springfield.

IOWA

Basic science examinations July 12 at the Capitol Bldg., Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

KANSAS

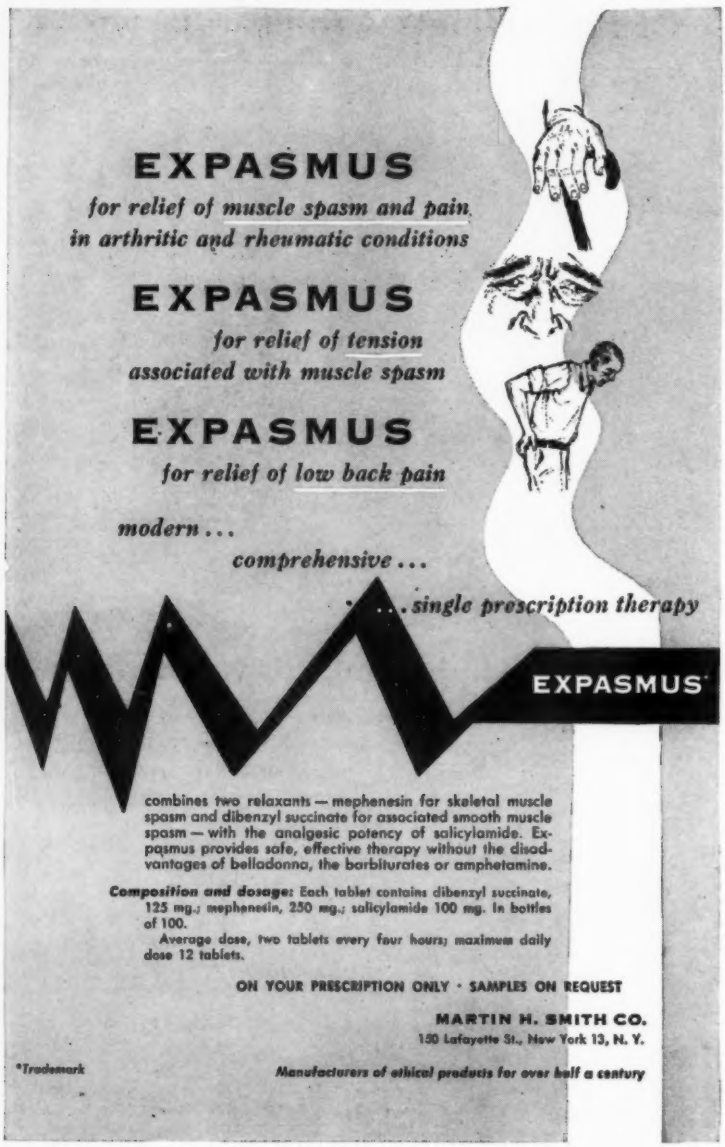
Examinations in June at Topeka. Address Forrest H. Kendall, D.O., secretary, Board of Osteopathic Examination and Registration, 420½ Pennsylvania Ave., Holton.

MAINE

Examinations June 14-15 at Augusta. Address George Frederick Noel, D.O., secretary, Board of Osteopathic Examination and Registration, Monument Square, Dover-Foxcroft.

MARYLAND

Examinations in June. Address Christopher L. Ginn, D.O., secretary, Board of Osteopathic Examiners, 419 No. Charles St., Baltimore 1.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MASSACHUSETTS

Examinations July 12. Address Robert C. Cochrane, M.D., secretary, Board of Registration in Medicine, Room 37, State House, Boston 33.

MINNESOTA

Basic science examinations June 7-8 at the University of Minnesota, Minneapolis. Applications must be filed by May 10. Address Raymond N. Bieter, M.D., secretary, Board of Examiners in the Basic Sciences, 105 Millard Hall, University of Minnesota, Minneapolis 14.

MISSISSIPPI

Examinations June 27-28 at the Robert E. Lee Hotel, Jackson. Applications must be filed by June 17. Address R. N. Whitfield, M.D., assistant secretary, Board of Health, Jackson.

NEVADA

Professional examinations June 25 at 210 W. Second St., Reno. Applications must be filed 2 weeks prior to examination. Address Walter J. Walker, D.O., secretary, Board of Osteopathic Examiners, 210 W. Second St., Reno.

Basic science examinations July 5. Address Donald G. Clooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.

NEW JERSEY

Examinations June 21. Address Patrick H. Corrigan, M.D., acting secretary, Board of Medical Examiners, 28 W. State St., Trenton.

NEW MEXICO

Basic science examinations July 17. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P. O. Box 1522, Santa Fe.

L. C. Boatman, D.O., Santa Fe, has been elected president of the Board of Examiners in the Basic Sciences for a term expiring in January, 1956.

NEW YORK

Examinations June 28-July 1. Address John W. Paige, M.D., chief, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

NORTH CAROLINA

Examinations July 1-2 at Raleigh. Reciprocity fee $25.00. No annual registration requirement. Address Frank R. Heine, D.O., secretary, Board of Osteopathic Examination and Registration, 926 Southeastern Bldg., Greensboro.

NORTH DAKOTA

Examinations July 9. Applications must be filed by June 15. Address G. L. Hamilton, D.O., secretary, Board of Osteopathic Examiners, Ringo Bldg., 119 S. Main St., Minot.

OHIO

Examinations June 13-15 at Men's Gymnasium, Ohio State University, Columbus. Address H. M. Platter, M.D., secretary, Medical Board, 21 W. Broad St., Columbus 15.

ONTARIO

Examinations June 23-25 at Toronto. Applications must be filed by May 14. Address D. Gordon Campbell, D.O., secretary, Board of Directors of Osteopathy, Drugless Practitioners Act, Province of Ontario, 2 Bloor St., E., Toronto 5.

OREGON

Examinations in July at Portland. Address Mr. Howard I. Bobbitt, executive secretary, Board of Medical Examiners, 609 Failing Bldg., Portland 4.

RHODE ISLAND

Professional examinations July 7-8. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

SOUTH CAROLINA

Examinations June 21 at Columbia. Address Ernest A. Johnson, D.O., secretary, Board of Osteopathic Examiners, Box 525, Summerville.

SOUTH DAKOTA

Professional examinations July 19-20 at Sioux Falls. Address Mr. John C. Foster, executive secretary, Board of Medical and Osteopathic Examiners, Room 300, First National Bank Bldg., Sioux Falls.

Basic science examinations first week in June. Address Gregg M. Evans, Ph.D., secretary, Basic Science Board, 310 E. 15th St., Yankton.

TENNESSEE

Examinations in July. Address M.

E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

TEXAS

Examinations June 20-22 in Fort Worth. Address M. H. Crabb, M.D., secretary, Board of Medical Examiners, 1714 Medical Arts Bldg., Fort Worth 2.

VERMONT

Examinations in June. Address Charles D. Beale, D.O., secretary, Board of Osteopathic Examination and Registration, Mead Bldg., Rutland.

WASHINGTON

Professional examinations July 10-13 at Seattle. Applications must be filed 15 days prior to examination. Address Mr. Edward C. Dohm, secretary, Professional Division, Department of Licenses, Olympia.

Basic science examinations July 6-7 at Seattle. Applications must be filed 15 days prior to examination. Address Mr. Dohm.

WEST VIRGINIA

Examinations June 27-28 at the Stonewall Jackson Hotel, Clarksburg. Applications must be filed 10 days prior to examination. A personal appearance 30 days prior to the meeting is necessary for licensing by reciprocity. Address W. S. Irvin, D.O., secretary, Board of Osteopathy, 202 Broadway St., Berkeley Springs.

WISCONSIN

Professional examinations July 12-13 at Milwaukee. Address Thomas W. Tormey, Jr., M.D., secretary, Board of Medical Examiners, State Office Bldg., 1 West Wilson St., Madison.

WYOMING

Examinations June 6 at Cheyenne. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

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Diagnosis: Fixed Essential Hypertension, Grade III

A. C.: Male, Negro, 31. Blood pressure 225/145, pulse rate 110. Excited, headaches, dizziness. Got good but not optimal reductions with Rautensin after 7 weeks, was therefore put on *combination therapy*—Rauvera, 4 tablets daily at 4-hour intervals after meals. (Each tablet contains 1 mg. mixed purified Rauwolfia alkaloids—the alseroxylon fraction—and 3 mg. mixed Veratrum alkaloids—alkavervir.) This accomplished a prompt additional reduction of blood pressure to 125/80 and of pulse rate to 84. Dizziness, headaches and excitability disappeared.

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REREGISTRATION OF OSTEOPATHIC LICENSES

During June—Hawaii, $5.00 for residents, $2.00 for nonresidents. Address Frank O. Gladding, D.O., secretary, Board of Osteopathic Examiners, 504 Kauikeolani Bldg., Honolulu 13.

Before June 30—Delaware, $20.00. Address Joseph S. McDaniel, M.D., secretary, Board of Medical Examiners, 229 S. State St., Dover.

June 30—Virginia, $1.00. Address K. D. Graves, M.D., secretary, Board of Medical Examiners, 631 First St., S. W., Roanoke.

July 1—Idaho, $10.00. Address D. W. Hughes, D.O., secretary, Board of Osteopathic Examiners, 203 Sun Bldg., Boise.

July 1—Kansas, $5.00. Address Forrest H. Kendall, D.O., secretary, Board of Osteopathic Examination and Registration, 420½ Pennsylvania Ave., Holton.

July 1—Michigan, $5.00. Address P. Ralph Morehouse, D.O., assistant secretary, Board of Osteopathic Registration and Examination, 214 So. Superior St., Albion.

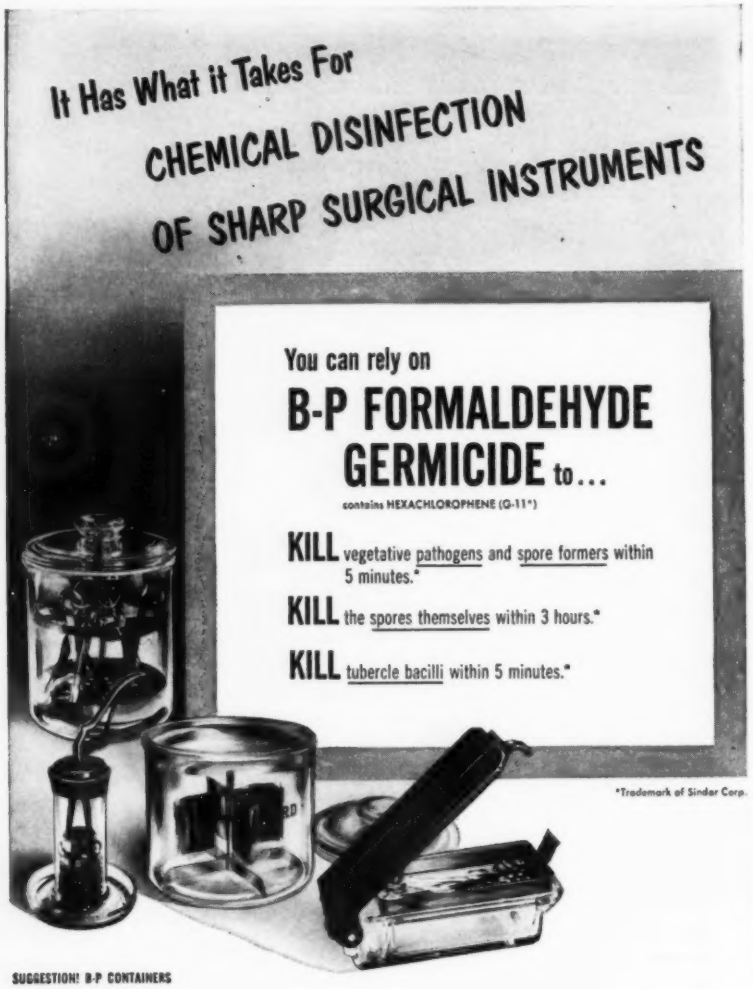
July 1—New Mexico, $5.00. Address H. E. Donovan, D.O., secretary, Board of Osteopathic Examiners, Ringo Bldg., tration, Donovan Osteopathic Hospital, Raton.

July 1—North Dakota, $3.00. Address G. L. Hamilton, D.O., secretary, Board of Osteopathic Examiners, Ringo Bldg., 119 S. Main St., Minot.

July 1—Oklahoma, $2.00. Address G. R. Thomas, D.O., secretary, Board of Osteopathy, 1100 N. W. 36th St., Oklahoma City 18.

July 1—West Virginia, $2.00. Address W. S. Irvin, D.O., secretary, Board of Osteopathy, 202 Broadway St., Berkeley Springs.

Within 60 days after July 1—Indiana, $5.00 for residents, $10.00 for nonresidents. Address Paul R. Tindall, M.D., secretary, Board of Medical Registration and Examination, 538 K. of P. Bldg., Indianapolis 4.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Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including hygiene, medical jurisprudence;

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene, medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

Part III is an oral and practical examination given under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles; therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

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Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

THE CHEMICAL CONTROL OF CANCER*

By James F. Holland, M.D.†

Satisfactory therapy for most patients with cancer has not been achieved. The advances in surgery which permit more extensive operations and the increased control of tumors which can be exerted by the newer techniques and more powerful equipment of modern radiotherapy still fall desperately short of solving the problem. Most physicians are legitimately skeptical that further advances in these two disciplines can successfully cope with the challenge.

In 1953 approximately 224,000 people died from cancer in the United States (*1*). Of all patients with malignant disease, it is probable that only one-fourth to one-third are cured today. Full use of present knowledge and better methods for earlier diagnosis would surely save more lives. There remains, however, millions of persons who will die from cancer in the foreseeable future despite the most informed use of surgical or radiothera-

*Reprinted from *Public Health Reports*, December, 1954.

†Dr. Holland, previously with the National Cancer Institute, Public Health Service, is now associate chief of medicine at Roswell Park Memorial Institute, Buffalo, N. Y.

peutic procedures, and regardless of the most meticulous attention to diagnostic techniques.

If an understanding of life is ever achieved in biochemical terms, it is a fair assumption that insight into the mechanisms of cancer will be realized pari passu. Despite the present lack of this understanding, two investigative approaches could be extended with reasonable hope of finding useful clinical applications.

The first approach, studies of carcinogenesis, may elucidate avoidable environmental or endogenous factors which, though now unknown, may be responsible for many cancer deaths. Recognition and avoidance of certain carcinogenic materials in the environment in the past have diminished their hazard. The observations of Pott in 1775 on carcinoma of scrotal skin in chimney sweeps (*2*) antedated by decades the appreciation of potential carcinogenicity associated with beta-naphthylamine and benzidine (bladder), ultraviolet radiation (skin), X-irradiation (skin, marrow, bone), and arsenic (skin), to name but a few well-established examples. Many other substances have been suspected of significant roles in human carcinogenesis: cigarette smoking (lung), urban soots (lung), chromate (lung), asbestos (lung), and smegma (penis and cervix) (*3-6*).

The positive identification of carcinogens is difficult. This difficulty may better be appreciated if it is recalled that the identity of the cancerous process in all its different types and loci is far from certain; there may be as many endogenous carcinogens as there are cancers, and perhaps multiple conditions must be satisfied for malignant growth to occur (*7, 8*). Despite the enormity of the task, the possibility is real that etiological and pathogenic factors can be identified and can then be avoided or counteracted. Research in carcinogenesis is deserving of the most intensive studies.

Chemotherapy of cancer is the second approach which has the potentiality of effecting a major decrease in cancer mortality. This potentiality has not been realized in man; indeed, no compound in use or under study at present offers significant promise of producing cure of human cancer. That there are systemic agents at all which alter the course of some malignant diseases is a signal achievement worthy of full exploitation. Several drugs in current use offer worthwhile benefit to cancerous patients. Close study of these agents may promote synthesis of drugs possessing cancerocidal rather than cancerostatic action, or discovery of compounds better able to provide remissions, if not cures.

Certain characteristics of the several cancer chemotherapeutic drugs permit their classification in different ways. Perhaps the broadest of these classifications describes the mechanism of action as interpreted clinically. Essentially these drugs may be segregated into (*a*) those agents which chiefly effect the host's response to, or toleration of, malignant tissue, with a variable degree of effect on the cancer cells, and (*b*) those agents which exert toxic action directly on cancer tissues, with a variable degree of toxicity in normal host cells.

The antineoplastic effect of the drugs to be discussed, although not cell-specific, is not diffuse nor wholly nonspecific. To a great extent the effect is limited to a certain disease or group of diseases. Without major exception, those malignant growths which currently can be favorably modified by drugs arise from hematopoietic tissues, lymphoid and reticular tissues, and from some of those organs whose functional status is influenced by endocrine control. For the vast majority of individuals with metastatic carcinomas, there is no drug of proven value. Many reasons might be advanced for the failure of response of carcinomatous cells. Surely there are chemical differences between carcinomatous and lymphomatous cells. These may involve such factors as the relative degree of variance from normal cells, the rate of growth, and the ability to tolerate or adapt to modified biochemical environment.

The drugs of clinical value with current applicability in cancer chemotherapy are: the nitrogen mustard compounds, mechlorethamine and triethylene melamine; the folic acid antagonists, a-methopterin and aminopterin; the purine analog, 6-mercaptopurine; the hormones, cortisone and testosterone; and the es-

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trogenic substance stilbestrol. Note will be made of adrenalectomy and hypophysectomy and of some chemical agents of limited clinical importance: urethane, myleran, and the ethylene phosphoramides.

NITROGEN MUSTARDS

Mechlorethamine (HN2) has usually been called nitrogen mustard, the generic name for the entire group of compounds, since its initial use in clinical medicine in 1942 (*9*). It is a highly active compound which forms stable linkages with many constituents of essential biochemical reactions.

Reactions occur with amino groups, carboxyl groups, sulfhydryl radicals, proteins, certain vitamins, purine and pyrimidine compounds, nucleic acids and with many other molecules of biological importance (*10*). The rate of chemical reaction with these compounds is extraordinarily rapid, and within several minutes the drug is fixed to tissue constituents. Most of the effects of such chemical alterations, however, are not manifest biologically until many hours or days later.

Triethylene melamine (TEM) possesses three ethylenimine groups and participates in reactions similar to those of mechlorethamine. There is no essential difference in their mechanisms of action, nor in their spectrum of antitumor effects. Both nitrogen mustard compounds demonstrate their greatest usefulness in the lymphomas, and each should be employed as a complement to radiotherapy. They do not supplant localized irradiation in the treatment of localized disease. When a lymphoma becomes "active" simultaneously in multiple sites, however, its control by radiotherapy is difficult or impossible. The delivery of sufficient radiation to several involved areas in an attempt to control tumor growth may induce profound marrow suppression or death.

The efficacy of HN2 and TEM is qualitatively the same in Hodgkin's disease and the lymphosarcomas. In the latter, the effects are usually the most striking in giant follicular lymphosarcoma, to a lesser extent in lymphocytic lymphosarcoma, and least in reticulum cell lymphosarcoma. In the course of Hodgkin's disease, fever, pruritis, exacerbations involving multiple node areas, and other indications of generalized disease are common. The administration of mechlorethamine or triethylene melamine is frequently propitious under such circumstances. Decrease or disappearance of tumor masses, beginning even within 24 hours, abolition of fever, relief from itching, and other evidences of clinical remission may follow shortly upon the administration of a nitrogen mustard compound.

The duration of such remission, if it occurs, is extremely variable; although in giant follicular lymphosarcoma remission of generalized disease may persist for months or years, the average duration in Hodgkin's disease and lymphocytic lymphosarcoma is measured in weeks. Radiotherapy may be used to great advantage to effect further regression in important local areas if the response to mechlorethamine or TEM has been suboptimal.

Another indication for mechlorethamine arises when space-occupying or invasive lymphomas have compressed to dangerous extent such organs as the trachea or the spinal cord. An initial vascular reaction with edema, frequently encountered after aggressive radiotherapy, is not seen with mechlorethamine. Intravenous administration of this compound may offer rapid effect in an emergency situation.

It has been stated that the nitrogen mustard compounds are effective in radioresistant lymphomas, that they preserve the sensitivity of a tumor to radiation, and that their use prolongs life in a way different from, and supplementary to, the benefit gained from radiotherapy. There would seem to be little substance to these claims. The apparent effect of nitrogen mustards in lymphomas not responding clinically to radiation is explained by the suppression of many areas of tumor activity simultaneously. In contrast, successive radiation treatments to local areas allow new or continuing tumor activity to flourish in untreated sites. When a tumor mass fails to regress locally under properly directed and adequate radiation therapy, no major regression is to be expected in that area after nitrogen mustard administration (*11*). It has been demonstrated that no difference in survival exists between a group of patients with Hodgkin's dis-

ease treated with radiotherapy alone and a group subjected alternately to courses of radiation and mechlorethamine treatment (*12*). The number of radiation treatment days per month in the mustard plus X-ray group was decreased, however, in contrast to radiation alone, thus affording easier clinical management of the patient.

In other diseases, nitrogen mustard compounds give measurable improvement, ranging from major regression of the neoplasm to symptomatic benefit. Because of the convenience of oral administration, TEM is profitably employed in mycosis fungoides, in chronic myelocytic leukemia, in chronic lymphocytic leukemia in small dosage, in metastatic cystadenocarcinoma of the ovary unsuited to other therapy, and in some cases if inoperable bronchogenic carcinoma (*13-15*). Recently, TEM has been found to produce far better results when used in conjunction with X-ray therapy, than X-ray alone in the treatment of retinoblastoma (*16*). There is no convincing evidence that the results of using the nitrogen mustards in sarcoidosis and the nephrotic syndrome, or in other nonmalignant diseases, justify the procedure.

The toxicity of mechlorethamine is expressed almost immediately in nausea and vomiting, but this manifestation is satisfactorily controlled in most individuals with chlorpromazine. Triethylene melamine is a less serious offender in this respect. Lymphocytopenia followed by granulocytopenia, thrombocytopenia, and anemia is common to both HN2 and TEM administration. The occurrence of severe marrow depression is not infrequent in those patients with marrow replacement by tumor or with suppression of hematopoiesis from prior radiation or chemotherapy. If tumor invasion of the marrow is decreased following the nitrogen mustards, however, hematopoietic improvement may counterbalance the pancytopenia from the drug (*13, 17*).

Mechlorethamine must be administered intravenously; this is conveniently performed by injecting the drug freshly dissolved in water or saline through the tubing of an infusion already running. Extravasation is highly irritating and venous thrombosis is not uncommon when HN2 is injected directly. The administration of 0.4 mg./kg. as a course of treatment is usually both safe and effective. This may conveniently be done by giving 0.2 mg./kg. on two successive evenings so that heavy nocturnal sedation may also be utilized to inhibit vomiting.

The parenteral administration of TEM would seem to command little advantage over HN2 (*13*). Its oral use, however, is worth while because hospitalizations solely for nitrogen mustard therapy can largely be avoided. When administered to a fasting patient, with 2 gm. of sodium bicarbonate to neutralize the gastric contents, formation of active ethylenimonium ion in acid medium is prevented, and reaction with compounds in the alimentary contents minimized. The use of 5 or 10 mg. of TEM initially, sometimes followed in 10 to 20 days with 5 and rarely 10 mg., has proved entirely satisfactory to achieve a "mustard effect" (*18*). The failure of repeated-dosing regimens (*13, 19*) to produce early hematopoietic depression, despite the large total doses ingested, probably lies in acidic activation and reaction of the ethylenimonium ion of TEM and consequent failure of absorption of fully active drug. When administered with sufficient alkali, it is likely that such large total doses would prove disastrous.

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FOLIC ACID ANTAGONISTS

The association of anemia in chicks with deficiency of dietary substances found in liver, spinach, and yeast was subsequently recognized to represent, in actuality, folic acid (pteroylglutamic acid) deficiency (*20-23*). Granulocytopenia in rats on purified diets was found to be correctible by administering folic acid (*24*). Acceleration of the acute leukemic process in children after administering derivatives of folic acid was described (*25*). The clinical trial of a compound chemically similar to folic acid, but antagonistic to it in a bacterial system dependent on folic acid for growth, was a normal sequence. The improvement in acute leukemia caused by the 4-amino substituted counterpart of folic acid, aminopterin, established the antineoplastic use of metabolic antagonists (*25*).

The 4-amino group is necessary for effective biochemical competition. A-methopterin (recently renamed methotrexate), the 4-amino-10-methyl-pteroylglutamic acid, has become the most widely used of

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the antagonists because of its clinical suitability. The folic acid antagonists affect many biochemical systems where transfer of single carbon units is involved. The syntheses of purines and nucleoproteins, the activities of several enzymes, the transformations of certain amino acids, and probably many other processes are diminished or interrupted (*26*).

In experimental systems involving mammalian tissues the inhibition exerted by a folic acid antagonist has been found to be irreversible by folic acid itself. Recent study has established, however, that folic acid exists in equilibrium with folinic acid in the body. This more active compound can successfully compete with aminopterin, and it has been suggested that the antagonist irreversibly prevents conversion of folic acid to folinic acid, whereas the antagonism to folinic acid is competitive and reversible (*27*).

In acute leukemia of childhood, the use of methotrexate may result in a temporary cessation of the clinical and morphologic characteristics of the disease. Within a period of 10 to 50 days, one may see disappearance of blast cells in the marrow and peripheral blood, regression of adenopathy and visceromegaly, reestablishment of erythropoiesis, normal leukocyte and platelet production, healing of bone lesions, and clinical well-being. Approximately one-third of those children treated will enjoy a complete or "hematologic" remission, and another one-fifth will sustain appreciable benefit despite the readily demonstrable presence of leukemic cells. The duration of improvement may continue for a few years in isolated instances, but in the average remission, benefit lasts about 10 weeks (*28*).

In the acute leukemias of adulthood, results comparable to those encountered in children are disappointingly rare. Although remissions do occur, only 13.5 percent of 163 adults selected from several reported series experienced any clinical benefit whatever (*28*).

Clinical and morphologic manifestations of acute leukemia reappear at some time during or after folic acid antagonist therapy. Although a second and even a third remission may sometimes be effected by reinstitution or increase of the drug, eventually the disease becomes refractory to folic acid antagonist therapy.

This refractoriness can be exemplified and interpreted by consideration of acute leukemia in rodents. The emergence of resistant strains of leukemic cells which can survive and multiply despite ordinarily suppressive levels of antagonist has been demonstrated (*29*). An understanding of the insensitivity of the cells to antagonists can probably be found in such factors as more efficient absorption and utilization of folic acid, failure of antagonist to gain access intracellularly, better detoxification of antagonist, or alteration of cellular metabolism so that different pathways of synthesis can be utilized when those dependent on folic acid or folinic acid are blocked (*30*). It is probable that eradication of billions of leukemic cells sensitive to an antagonist leaves a residue of preexistent mutant cells insensitive to the drug. When multiplication of these unopposed mutants produces a nearly pure strain of resistant cells, no response to the provoking antagonist can be anticipated. Such insensitivity to folic acid antagonists in leukemic cells is a heritable trait, and strains of transplantable rodent leukemia which have developed resistance are maintained in many laboratories. A similar explanation is probably applicable to human acute leukemia when refractoriness to a metabolic antagonist occurs.

The chronic leukemias are relatively resistant to the folic acid antagonists, and their use in these diseases finds little support in the clinical literature. The temporary response of neuroblastomas in children to methotrexate administration occurs sufficiently often to make this a reasonable palliative effort. Sporadic and unimpressive response has been reported in a variety of other neoplastic diseases investigated, but in none of these are the results sufficiently encouraging to suggest clinical application.

Toxicity is most apparent in those body tissues where rapid cellular proliferation is characteristic. Marrow depression with severe leukopenia, granulocytopenia, anemia, and thrombocytopenia may occur. The epithelium of the alimentary canal is highly susceptible.

Anorexia, vomiting, diarrhea, and abdominal pain are often early indications of host toxicity. Ulceration of the oral mucosa, necrosis of interdental papillae and intestinal bleeding are more serious adverse effects. Skin eruptions and alopecia are occasionally seen.

The toxicity of aminopterin or methotrexate is counteracted to some extent by the use of folinic acid (citrovorum factor, Leucovorin). The biochemical competition exerted by folinic acid interrupts the metabolic blockade of the antagonist, and normal chemical pathways are presumably reestablished. A feeling of well-being replaces the malaise and apathy which accompany the toxic state. Remission of the signs of toxicity follows. There is no conclusive evidence that folinic acid per se modifies the acute leukemia (*31*).

Methotrexate is administered in doses of 2.5 to 5.0 mg. daily, orally. Only rarely are larger daily doses worth while, and then the risk of fulminant toxicity is considerable. The minimum effective dose of folinic acid for relief of methotrexate toxicity is not firmly established. In the range from 10 to 30 mg. daily in divided intramuscular doses, satisfactory results have been observed (*31*).

PURINE ANALOGS

The dominant position that nucleoproteins occupy in cellular metabolism and reproduction has prompted extensive studies of their biochemistry. Nucleic acid is the highly polymerized residue of nucleoprotein after protein has been removed. The constituent moieties of any particular nucleic acid are molecules of phosphoric acid, of a pentose sugar, ribose or desoxyribose, and of a few types of the heterocyclic compounds, pyrimidines and purines. Analogous compounds to the pyrimidine and purine bases have been synthesized (*32, 33*) in search of metabolic antagonists which could modify the formation of nucleoproteins in cell reproduction and survival. Many antimetabolites active against micro-organisms, malarial parasites, and animal tissues have resulted from these systematic studies.

The activity of 6-mercaptopurine against certain transplantable animal tumors has been shown to be remarkably efficient (*34, 35*). Its introduction into clinical medicine in April 1952 was rapidly followed by a definition of its scope of usefulness; to date it is the most practicable of the purine analogs for use in man.

The mechanism of action of 6-mercaptopurine is unknown. It has not yet been proved that 6-mercaptopurine is incorporated into nucleic acids, although much evidence suggests it. Further, if incorporated, it is not known exactly how this is detrimental to the cell. There are physiological compounds of importance other than nucleic acids which contain adenine, such as coenzymes, and it is possible that 6-mercaptopurine exerts antagonism to adenine in these compounds.

Clinically, 6-mercaptopurine demonstrates salutary effects in the acute leukemias and in chronic granulocytic leukemia (*36*). Approximately one-third of the children and a smaller proportion of adults with acute leukemia enjoy major reduction in acute leukemic pathology and symptoms when taking 6-mercaptopurine. A lag period of a few days to several weeks may precede response. When well established, a remission may progress to disappearance of adenopathy, visceromegaly, and abnormal cells in marrow aspirates. The duration of remissions is usually measured only in weeks or months. Relapse occurs when blast suppression is ineffective. This is explained, as with the folic acid antagonists, by the resistance of the leukemic cells to the biochemical actions of 6-mercaptopurine. Similar resistance phenomena have been demonstrated and studied in murine leukemia (*37*).

In chronic granulocytic leukemia, satisfactory decrease in peripheral white count and visceromegaly can be achieved with 6-mercaptopurine. In the blastic crisis of the acute preterminal phase of chronic granulocytic leukemia, reports of temporary improvement from 6-mercaptopurine have been made. This stage of the disease has been singularly unresponsive to radiation or other chemical agents, and 6-mercaptopurine commands a unique advantage here. In generalized reticulum cell lymphosarcoma, 6-mercaptopurine has produced minor benefit. It holds no significant value for chronic lymphocytic

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leukemia, the other lymphomas, or in patients with metastatic carcinoma (*36*).

In both experimental and clinical leukemia it has been shown that the inhibitions effected by folic acid antagonists and purine analogs are not biochemically the same. Thus, resistance to an antifolic compound does not preclude sensitivity and response to an antipurine, and vice versa (*36, 37*). The sequential use of these compounds, therefore, offers the possibility of two remissions. There are theoretical advantages to the simultaneous use of both compounds from the start of treatment (*38*), and this area is being explored clinically (*39*).

The toxicity of 6-mercaptopurine is manifest by phenomena quite similar to those of the antifolic compounds. Substantial inhibition of hematopoiesis with resultant pancytopenia may lead to death. In some instances, leukopenia precedes thrombocytopenia and anemia sufficiently to afford warning of impending danger. Gastrointestinal dysfunction occurs less frequently than with methotrexate, but may be equally as severe. Oral ulceration ascribed to the drug has occasionally been seen in patients with acute leukemia. No satisfactory agent for the reversal of the toxicity of 6-mercaptopurine has been used in man. In rodents, several compounds which already contain adenine, such as adenylic acid and adenosine, or guanine, such as guanylic acid and guanosine, are effective in diminishing the effects of 6-mercaptopurine. The purine bases, adenine, guanine, and hypoxanthine, are inferior an allaying toxicity (*40*).

The dose of 6-mercaptopurine ordinarily employed is 2.0 to 3.0 mg./kg. daily by mouth. More intensive dosage regimens are said to offer slightly more efficacy, but the toxicity encountered is increased. There is little support for intermittent dosage regimens. Once the drug has been instituted in acute leukemia, discontinuation, except for toxicity or refractoriness, is unnecessary. Sufficient time has not elapsed to judge the optimal methods for use of 6-mercaptopurine in chronic granulocytic leukemia except in its acute terminal phase where continuous administration is practiced.

THE ADRENAL CORTICAL STEROIDS

The adrenal cortical steroids, cortisone and hydrocortisone produce qualitatively indistinguishable responses and will not be treated separately. Corticotrophin elicits entirely similar phenomena by inducing endogenous corticosteroid production. Accordingly, cortisone will be used for the generic name.

The response to cortisone has been widely studied in patients with malignant disease. Some apparently specific responses occur in addition to a sense of physical well-being, polyphagia, and elation, which many patients may experience temporarily irrespective of the type of their neoplasm. The most satisfactory specific effect is seen in acute leukemia of childhood. With dramatic speed, regression of the signs of disease may occur when cortisone is administered. The swiftness, and to some extent the surety, make cortisone the agent of choice when beginning treatment of a critically ill acute leukemia patient. Cessation of hemorrhage, often before any change in platelet count, and return toward normal hematopoiesis may be evident within a few days. Complete regression of the disease state may follow.

The mechanism of action is unknown. There is a specific effect on the leukemic cell over and beyond the systemic benefits, which is probably akin to the lymphocytolysis seen in normals. The remissions induced are shorter than those from a-methopterin or 6-mercaptopurine, averaging about 6 weeks. If relapse appears while taking adequate cortisone, increase in dosage is but a temporary expedient, and the disease rapidly becomes wholly uninfluenced by the drug. It is in this period of increasing dose and declining antineoplastic activity that the patient may be subjected to intense hyperadrenocorticism.

(To be concluded in the June JOURNAL*)*

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GAINS IN LONGEVITY SINCE 1850*

For more than a century, the average length of life in our country has been steadily increasing. Under mortality conditions prevailing in 1850, the expectation of life at birth was less than 40 years. By 1954, the figure had risen to about 70 years. Thus, at least 30 years have been added to the expectation of life at birth because of the reduction in mortality since the middle of the last century.

The gains in longevity have been much greater in the period since the turn of the century than they were in the preceding 50 years. Among white males, for example, the increase in the expectation of life at birth was about 10 years from 1850 to 1900-1902, and about 18½ years in the following half century; among white females the corresponding increases were 10½ years and 21½ years. Progress in extending the average length of life was accelerated with the quickened tempo of advances in environmental sanitation and in preventive and curative medicine. In the latter part of the 19th century, the infectious diseases were still taking a heavy toll of life annually, and at times epidemics virtually ravaged sections of the population. For instance, in 1891 the death rate from typhoid fever in Chicago reached 172.7 per 100,000 population. Similarly, the diphtheria death rate in Baltimore was 260.5 per 100,000 in 1881-1882. Both these diseases have been practically wiped out as causes of death not only in our cities but in the country as a whole. There has been no major epidemic in the United States for more than 35 years, the last one being the influenza pandemic of 1918-1919.

Although the gains in the expectation of life have been largest in childhood and early adult life, some progress is observed even at the older ages. Since 1901, the expectation of life at age 40 has increased about four years among white men and nearly seven years among white women; at age 65 the corresponding gains have been 1½ years and three years. Currently, white men at age 65 can look forward to about 13 years on the average, and white women to 15½ additional years of life. At all ages, the increase in longevity has been greater for females than for males.

In 1952, the expectation of life at birth for white females exceeded that for white males by six years. The sex difference diminishes with advance in age, but it still amounts to three years at age 60. Mortality among white females has dropped to remarkably low levels prior to midlife. The death rate among them is now less than 1 per 1,000 in the age range 3-28, and remains under 2 per 1,000 through age 38. Among white males, death rates under 1 per 1,000 are recorded at ages 4-14, and stay under 2 through age 32.

The nonwhite population likewise has made considerable gains in longevity since the turn of the century. The ex-

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pectation of life at birth for nonwhite males increased 26½ years from 1900-1902 to 1952; for nonwhite females the corresponding rise was 28⅔ years. During this period the nonwhite population has made more rapid progress than the white population, thus reducing the difference in longevity between the two groups. Nevertheless, the expectation of life at birth for nonwhites in 1952 was still well below that for the white population—by 7½ years among males and by almost 9 years among females.

The extraordinary progress made in prolonging life during the past century reflects, in the main, the effective control gained over the diseases of environmental origin—tuberculosis, the common infections of childhood, pneumonia, and many others. Future gains may be more difficult to achieve because they will depend largely upon reducing the toll from diseases and conditions which, in the present state of knowledge, are for the most part of obscure or unknown etiology. These include many of the cardiovascular-renal diseases, the cancers, and the diseases of early infancy. Yet there is every reason to believe that the toll of premature death can be reduced further as the medical and sanitary sciences continue to develop new knowledge and make more effective use of what is already known.

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DECLINE IN MORTALITY FROM GALLBLADDER DISEASES*

The striking reductions in mortality from the major infectious diseases have tended to overshadow the large gains made in the control of many other diseases. Gallbladder disorders, which affect a very considerable proportion of the adult population, are a good example of the latter.

Among white females insured under Industrial policies in the Metropolitan Life Insurance Company, the death rate from the diseases of the gallbladder (including gallstones) declined more than three fifths in the 15 years between 1936-1938 and 1951-1953—from 7.7 to 2.9 per 100,000. Among white males, the death rate was cut in half—from 3.4 to 1.7 per 100,000.

Policyholders in every age group shared in the improvement in mortality from gallbladder conditions, with the largest relative reductions in the 15-year period being recorded at the ages under 55. Thus, at ages 35-44 the decrease was four fifths among white women and two thirds among white men. Even at ages 65-74, where the death rates are highest in this insurance experience, the reduction for the two sexes exceeded 40 percent and 25 percent, respectively. As a result of the more rapid improvement among women, the sex difference in the mortality from these diseases diminished appreciably. Whereas in 1936-1938 the death rate among females in the age range 35-74 exceeded that among males by about 125 percent, in 1951-1953 the excess was only 60 percent.

The reduction in mortality from gallbladder disorders may be attributed to a number of factors. Of particular importance for the large proportion of patients coming to operation has been the decrease in surgical mortality resulting from advances in operative technique, the control of infection by chemotherapy and antibiotics, and the prevention of surgical shock and pulmonary embolism. In one large clinical experience (Mayo Clinic), the surgical mortality rate for removal of the gallbladder dropped nearly two thirds in about a decade—from 1.9 percent in 1939-1941 to .7 percent in 1950-1952. Patients with gallbladder diseases treated without surgical intervention have benefited materially from improved regulation of diet and better medical care generally.

A long-term follow-up study of insured lives with a history of gallbladder disorders indicated that such persons have a relatively favorable mortality. The excess mortality was smallest among per-

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sons with a history of the disease who, up to the time they were insured, had received only medical treatment for the condition. The excess was less for those with a single attack than for those with recurrent attacks. Moreover, the mortality decreased as the time elapsed since attack increased. Thus, persons who had had a single attack within two years prior to application for insurance had a mortality nearly one and a third times that of standard risks, but the mortality was only slightly above the average when the time elapsed since attack was longer than two years. Among medically treated cases which had experienced recurrent attacks, the mortality was about one and a third times that of standard risks.

Persons with a history of removal of the gallbladder within five years prior to application for insurance had a mortality about one and a quarter times that of standard risks, or somewhat higher than that among medically treated cases. For those accepted for insurance more than five years after operation, the mortality was about the same as in the corresponding group of medically treated cases. The results were less favorable for those with a history of drainage of the gallbladder than for persons who had had their gallbladder removed. Among the former, the mortality for cases insured within five years of operation was about one and a half times that of standard risks. The generally higher mortality among persons with a history of surgery for gallbladder disease reflects, in part at least, the fact that operated cases were generally the more severe. The relatively poor experience among those with a history of drainage reflects the circumstances which made the more radical operation inadvisable. Among the surgically treated cases the excess mortality was due primarily to complications of the disease, whereas among the medically treated cases it was the result of recurrent attacks.

ALL-TIME LOW MORTALITY IN 1954*

Mortality among the Industrial policyholders of the Metropolitan Life Insurance Company, continuing its long-term downward trend, set a new low record in 1954 with a death rate of 6.2 per 1,000 for all ages combined. This compares with the rate of 6.5 for 1953, and represents a reduction of nearly one fifth since 1940 and of one half since 1911. These decreases actually understate the improvement, because the proportion of policyholders at the older ages, where the death rates are highest, has been increasing. When allowance is made for this factor, the decrease in mortality among the Industrial policyholders between 1911 and 1954 comes to nearly two thirds.

The splendid health record for 1954 was due in part to the freedom from major outbreaks of respiratory disease, such as occurred in the early months of 1953. In larger measure, the unprecedented low death rate reflects the remarkable advances made in the medical sciences and public health administration, and the high standard of living prevailing among American wage-earners and their families.

Each of the major causes of death, except the malignant neoplasms, and virtually all the causes of lesser importance registered a lower mortality rate in 1954 than in 1953. Among the causes of death establishing new lows last year were tuberculosis, pneumonia, influenza, appendicitis, syphilis, the complications of pregnancy and childbearing, suicide, and homicide.

During 1954 there were 113,000 deaths reported among the Company's Industrial policyholders. If the death rates by age in each color-sex group had been the same as those prevailing in 1911, there would have been 304,000 deaths among these insured. Thus, about 191,000 deaths were postponed in 1954 because of the improvement in mortality since 1911.

MORTALITY BY SEX AND AGE

White policyholders in every age group recorded a lower death rate in 1954 than

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*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, January, 1955.

in 1953. The largest relative decreases in each sex were at the younger ages, which conforms to the pattern observed over the years. Among white males, the reduction in mortality between 1911-1915 and 1954 ranged from more than 90 percent at ages 1-4 to about 40 percent at ages 55-74; white females showed an even more favorable experience.

MAJOR CAUSES OF DEATH

The death rate from the cardiovascular-renal diseases dropped 5 percent between 1953 and 1954—from 341.6 to 324.8 per 100,000 policyholders. Each of the major diseases in this group showed a decrease in mortality; even the death rate from coronary artery disease fell from 108.7 to 104.8 per 100,000. The appreciable decline in the mortality from the cardiovascular-renal diseases may have been associated with the fact that the influenza epidemic which struck early in 1953 was not repeated in 1954. Outbreaks of respiratory disease usually hasten the death of many people with chronic illnesses.

In contrast to the trend for the other major causes of death, the malignant neoplasms recorded an increase in death rate from 126.7 per 100,000 in 1953 to 128.7 in 1954. Malignancies of the digestive system and of the respiratory system each showed a rise in mortality of slightly more than 4 percent.

Nearly three fourths of all the deaths among the Industrial policyholders last year were attributed to the cardiovascular-renal diseases or to the malignant neoplasms. The first group alone accounted for more than one half the total mortality; the malignancies ranked second with an additional one fifth of the deaths.

Despite a sharp drop in the recorded mortality from diabetes resulting from recent changes in classification procedures, this disease is by no means a minor item in the list of causes of death. Last year the diabetes death rate was 14.9 per 100,000, slightly under the rate of 15.6 for 1953.

COMMUNICABLE DISEASES

Each year witnesses new and striking victories scored against tuberculosis. Among the Company's Industrial policyholders the death rate from the disease in 1954 fell to the unprecedented low of 8.9 per 100,000, from the previous minimum of 10.4 established the year before. The tuberculosis death rate has dropped by one half in three years and by three quarters in a decade. It is rather startling to recall that the mortality from this disease in 1911 was no less than 224.6 per 100,000, or 25 times that for last year. There appears to be little doubt that further progress will be made in the control of tuberculosis in the years immediately ahead.

Exceptionally favorable, too, was last year's record for pneumonia and influenza. The death rate from the two diseases combined decreased more than one fifth between 1953 and 1954. For pneumonia alone, the death rate dropped from 15.1 to 12.9 per 100,000, or 15 percent, while for influenza the reduction was from 2.7 to 1.0 per 100,000; both diseases established a new low mortality in 1954. It should be pointed out, however, that these diseases still constitute a major medical and public health problem. Some forms of respiratory disease do not respond to the antibiotics or other specific therapy, and for these effective immunization procedures are still lacking; moreover, there is no assurance that a virulent epidemic will not strike again, as it has in times past. On the brighter side, significant progress has been made in developing an effective vaccine against certain types of influenza.

The death rate from acute poliomyelitis was .7 per 100,000 policyholders in 1954, which compared with 1.0 the preceding year and with 1.5 in 1952. However, the number of cases of the disease reported in the general population of the United States during 1954 (about 38,750) was 8 percent greater than in 1953.

The mortality from the principal communicable diseases of childhood—measles, scarlet fever, whooping cough, and diphtheria—continued at the remarkably low rate of .3 per 100,000 Industrial policyholders. Little more than a decade ago, the death rate was 10 times as high. In 1911, the mortality from diphtheria alone was at least 90 times the present rate for all four diseases combined.

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The death rate from complications of pregnancy and childbirth among the Industrial policyholders decreased to a new minimum of 1.1 per 100,000 in 1954, or to a level 15 percent below that for the year before. This improvement occurred despite an increase in the number of births in the United States to a new high of more than 4,000,000. In 1954, the mortality incidental to maternity among the insured was only about one half that five years ago and little more than one quarter the rate 10 years ago.

OTHER RECORD LOWS

Two other diseases which recorded new low death rates in 1954 merit special mention. The mortality from syphilis was down to 2.4 per 100,000 in 1954, or two fifths below that four years earlier. For appendicitis the death rate was reduced from 1.8 per 100,000 in 1950 to 1.3 in 1954. The control gained over the mortality from both these diseases is attributable in large measure to the use of the antibiotics.

EXTERNAL CAUSES OF DEATH

Accident fatalities in this insurance experience decreased from 38.6 to 36.8 per 100,000 between 1953 and 1954. Motor vehicle, home, and occupational (civilian) accidents all shared in the improvement. The death rate from motor vehicle accidents decreased from 16.3 per 100,000 in 1953 to 14.6 in 1954, the lowest rate since 1949.

Both suicide and homicide dropped to all-time low levels in 1954, the rates being 5.5 and 2.3 per 100,000, respectively.

EXPECTATION OF LIFE

The expectation of life at birth among the Company's Industrial policyholders, based on provisional mortality data for 1954, reached a new high of 69.9 years. This means that if these insured experienced the mortality rates of 1954 throughout life they would live 69.9 years on the average. Actually, the average will probably be higher inasmuch as further reductions in mortality may be expected. The figure for expectation of life at birth in 1954 was one year greater than that for 1953, and five and a half years above that of a decade ago.

FAVORABLE OUTLOOK FOR THE FUTURE

There is good reason to expect that the mortality and longevity of American wage-earners and their families will improve further. Medical and public health services continue to grow both in number and quality, while intensified research is constantly adding to the store of knowledge on the prevention, diagnosis, and treatment of disease.

Books Received

CURRENT THERAPY 1955. Latest Approved Methods of Treatment for the Practicing Physician. Edited by Howard F. Conn, M.D. Cloth. Pp. 692. Price $11.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1955.

NEUROLOGY. Vols. 1, 2, and 3. By S. A. Kinnier Wilson, M.A., M.D., D.Sc. (Edin.), F.R.C.P., Formerly Physician, National Hospital, Queen Square; Senior Neurologist, King's College Hospital; Consulting Neurologist, Metropolitan Asylums Board (L.C.C.); Officer de L'Instruction Publique, R.F.; Honorary Fellow, Royal Academy of Medicine, Turin; Honorary Member, Royal Academy of Medicine, Belgium, National Academy of Medicine, Rio de Janeiro, Neurological Societies of Italy, Poland, Denmark, Holland, Brazil, Paris, Vienna, New York and Philadelphia, The Japanese Association of Psychiatry and Neurology, the Society of German Neurologists, the Medical Society of Copenhagen and the American Neurological Association; Corresponding Member, Neurological Society of Warsaw. Ed. 2. Cloth. Pp. 2060, with illustrations. Price $37.50. The Williams & Wilkins Company, Mount Royal & Guilford Aves., Baltimore 2, 1955.

BLOOD GROUPS IN MAN. By R. R. Race, Ph.D. (Cambridge), M.R.C.S. (England), F.R.S., Director, Medical Research Council Blood Group Research Unit, The Lister Institute, London; and Ruth Sanger, Ph.D. (London), B.Sc. (Sydney), Medical Research Council Blood Group Research Unit, The Lister Institute, London. Ed. 2. Cloth. Pp. 400, with illustrations. Price $7.50. Charles C Thomas, Publisher, 301-327 East Lawrence Ave., Springfield, Ill., 1954.

Financing Hospital Care in the United States, Volume 3: FINANCING HOSPITAL CARE FOR NONWAGE AND LOW-INCOME GROUPS. Edited by Harry Becker. Cloth. Pp. 110, with illustrations. Price $2.50. McGraw-Hill Book Company, 330 W. 42nd St., New York 36, 1955.

THE PHILOSOPHY OF X. By Paul Doremus. Cloth. Pp. 98. Price $2.50. Comet Press Books, 11 West 42nd St., New York 36, 1954.

HEMATOLOGY. By Cyrus C. Sturgis, M.D., Professor of Internal Medicine, Chairman of the Department of Internal Medicine, University of Michigan Medical School; and Director of the Thomas Henry Simpson Memorial Institute for Medical Research, University of Michigan, Ann Arbor, Michigan. Ed. 2. Cloth. Pp. 1222, with illustrations. Price $19.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1955.

CLINICAL DISORDERS OF HYDRATION AND ACID-BASE EQUILIBRIUM. By Louis G. Welt, M.D., Professor of Medicine, Department of Medicine, University of North Carolina. Cloth. Pp. 262, with illustrations. Price $6.00. Little, Brown & Company, Boston 6, 1955.

TEXTBOOK OF THE RHEUMATIC DISEASES. Edited by W. S. C. Copeman, O.B.E., M.D., F.R.C.P., Senior Physician, Arthur Stanley Institute for Rheumatic Diseases, Middlesex Hospital; Physician in charge of Department of Rheumatic Diseases, West London Hospital; Chairman, Empire Rheumatism Council; and late President, European League against Rheumatism. Ed. 2. Cloth. Pp. 754, with illustrations. Price $11.00. The Williams & Wilkins Company, Mount Royal & Guilford Aves., Baltimore 2, 1955.

FLUOROSCOPY IN DIAGNOSTIC ROENTGENOLOGY. By Otto Deutschberger, M.D., Assistant Clinical Professor of Radiology, New York Medical College; Roentgenologist in charge, Bird S. Coler Memorial Hospital, New York; Associated Visiting Roentgenologist, Metropolitan Hospital, New York. With an introduction by Frank J. Borrelli, M.D., F.A.C.R. Cloth. Pp. 771, with illustrations. Price $22.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1955.

ION EXCHANGE AND ABSORPTION AGENTS IN MEDICINE. The Concept of Intestinal Bionomics. By Gustav J. Martin, Sc.D., Research Director, The National Drug Company, Philadelphia. Cloth. Pp. 333, with illustrations. Price $7.50. Little, Brown & Company, 34 Beacon St., Boston 6, 1955.

BRITISH OBSTETRIC AND GYNECOLOGICAL PRACTICE. Vols. I and II. Edited by Sir Eardley Holland, M.D. (Lond.), F.R.C.P., F.R.C.S., F.R.C.O.G., Hon. M.D. (Dublin); Hon. LL.D. (Birmingham); Hon. LL.D. (Leeds); Hon. F.R.C.S. (Edin.); Consulting Obstetric and Gynecological Surgeon, London Hospital; Late President, Royal College of Obstetricians and Gynecologists; President XIIth British Congress (1952); Hon. Fellow American Gynecological Society; Corresponding Member, French and German Gynecological Societies; and Aleck Bourne, M.A., M.B., B.Ch. (Cantab.), F.R.C.S., F.R.C.O.G.; Consulting Gynecologist, St. Mary's Hospital, London; Consulting Obstetric Surgeon, Queen Charlotte's Hospital; Consulting Surgeon, Samaritan Hospital for Women, London. Cloth. Pp. 2022, with illustrations. Price $29.50. F. A. Davis Company, 1914 Cherry St., Philadelphia 3, 1955.

PRIMARY ANATOMY. By H. A. Cates, M.B., Late Professor of Anatomy and Director of the School of Physical and Health Education, University of Toronto; and J. V. Basmajian, M.D., Associate Professor of Anatomy, University of Toronto. Ed. 3. Cloth. Pp. 339, with illustrations. Price $5.75. The Williams & Wilkins Company, Mount Royal & Guilford Aves., Baltimore 2, 1955.

Christopher's MINOR SURGERY. Edited by Alton Ochsner, M.D., F.A.C.S., William Henderson Professor of Surgery and Chairman of the Department of Surgery, Tulane University of Louisiana School of Medicine; and Michael E. DeBakey, M.D., F.A.C.S., Professor of Surgery and Chairman of the Department of Surgery, Baylor University College of Medicine. Ed. 7. Cloth. Pp. 547, with illustrations. Price $9.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1955.

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THE JOINTS OF THE EXTREMITIES. A Radiographic Study. By Raymond W. Lewis, M.D., Formerly Director, Department of Radiology, Consultant in Roentgenology, The Hospital for Special Surgery, New York, New York. Cloth. Pp. 108, with illustrations. Price $8.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1955.

THE MEDICAL CARE OF THE AGED AND CHRONICALLY ILL. By Freddy Homburger, M.D., Research Professor of Medicine, Tufts College Medical School. Cloth. Pp. 253, with pen and ink sketches by Raoul Dufy. Price $5.75. Little, Brown and Company, 34 Beacon St., Boston 6, 1955.

A TEXTBOOK OF PHYSIOLOGY. Edited by John F. Fulton, M.D., Sterling Professor of the History of Medicine, Yale University School of Medicine. Ed. 17. Cloth. Pp. 1275, with illustrations. Price $13.50. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1955.

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1. Cass, L. J. and Frederik, W. S.: Malt Soup Extract as a Bowel Content Modifier in Geriatric Constipation. Journal-Lancet, 73:414 (Oct.) 1953.

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CHANGES OF ADDRESS AND NEW LOCATIONS

Adams, John E., KC '54; Doctors Hospital, 1087 Dennison Ave., Columbus 1, Ohio

Alvarado, Miguel M., from Chicago, Ill., to Grandview Hospital, 405 Grand Ave., Dayton 5, Ohio

Angellotti, Robert L., KC '54; 3770 Demean, Melvindale, Mich.

Atterberry, Kenneth Dale, from Foster Bldg., to Camdenton Clinic & Hospital, Camdenton, Mo.

Babcock, Robert E., from North Hollywood, Calif., to 9311 E. Whittier Blvd., Whittier, Calif.

Bachrach, Richard M., PCO '54; 4521 Spruce St., Philadelphia 39, Pa.

Barnes, Forrest H., DMS '54; 4403 Adams St., Kansas City 3, Kans.

Baumgaertel, G. George, Jr., PCO '54; 6606 N. 12th St., Philadelphia 26, Pa.

Bean, Roger A., from Springfield, Ohio, to 31 N. Elm St., West Carrollton, Ohio

Becker, Alan Harvey, CCO '54; 18703 Woodingham Drive, Detroit 21, Mich.

Beckham, Alfred, Jr., from 10812 S. Flallon Ave., to 12037 Elmcroft, Norwalk, Calif.

Beeson, J. Raymond, from Hendrick Bldg., to 1156 Boulevard Way, Box 605, Walnut Creek, Calif.

Bennett, Roger E., from 203 S. Broad St., to 1610 Gage Drive, Middletown, Ohio

Bentz, Robert L., PCO '54; Bay View Hospital, 23200 Lake Road, Bay Village, Ohio

Billings, William S., from Philadelphia, Pa., to 200 Haddon Ave., Haddonfield, N. J.

Black, John Russell, from Miami, Fla., to 8890

S. W. 96th St., South Miami 43, Fla.
Black, Linnie K., from Miami, Fla., to 8890 S. W. 96th St., South Miami 43, Fla.
Bohrer, Omer L., from Commercial St., to 117 N. Jefferson St., Lebanon, Mo.
Boettger, Lloyd, from Camarillo, Calif., to 2471 Waterloo Road, Stockton 5, Calif.
Boone, Richard F., from Big Spring, Texas, to 2215 Carson St., Fort Myers, Fla.
Bragg, Jackson B., KCOS '54; Laughlin Hospital & Clinic, 711-15 W. Jefferson St., Kirksville, Mo.
Brill, Jack J., from Amherst, Ohio, to 4625 Broadway, Lorain, Ohio
Buehler, John L., from 6014 S. Western Ave., to 968 W. Vernon Ave., Los Angeles 37, Calif.
Cable, Guy W., from Oran, Mo., to Bismarck, Mo.
Cammitz, Leonard, from 9345 Rutherford, to 15373 Kentfield, Detroit 23, Mich.
Carlin, Edwin S., from Media, Pa., to 718 W. Springfield Road, Springfield, Pa.
Carlson, Edward A., from Box 692, to Box 146, San Andreas, Calif.
Claus, Leslie V., from Los Angeles, Calif., to 4116 E. Compton Blvd., Compton, Calif.
Clifton, Arthur L., from Freeland, Mich., to 128 N. Ross, Beaverton, Mich.
Cook, Glenn E., PCO '54; Box 146, New Lebanon, Ohio
Couts, Fred A., from Overland, Mo., to Lackland Clinic, 2335 Brown Road, St. Louis 14, Mo.
Crabb, Samuel L., from Chula Vista, Calif., to 4306 40th St., San Diego 16, Calif.
Craven, Douglas K., from Alhambra, Calif., to 228 N. Garfield Ave., Monterey Park, Calif.
Cunningham, E. Jane, from Flint, Mich., to Paradise Bay Trailer Park, Bradenton, Fla.
Davis, E. Paul, KCOS '54; 504 S. Ely St., Kirksville, Mo.
Dean, W. E., from Bozeman, Mont., to 2004 Peninsula Drive, Moses Lake, Wash.
DeLille, George A., from 325 W. Jefferson Blvd., to 3200 W. Sixth St., Los Angeles 5, Calif.
DePrisco, John, PCO '54; Allentown Osteopathic Hospital, 1746 Hamilton St., Allentown, Pa.
Dooley, Wayne, from 609 S. Grand Ave., to 6005 Melrose Ave., Los Angeles 38, Calif.
Dubin, H. Mayer, from Los Angeles, Calif., to 8335 E. Second St., Downey, Calif.
Ehlinger, Norman J., from Box 432, to 125 N. Canal St., Canal Fulton, Ohio
Eske, Louis H., Jr., DMS '54; Grandview Hospital, 405 Grand Ave., Dayton 5, Ohio
Farrell, Leslie E., KC '54; Waldo General Hospital, 8511 15th Ave., N. E., Seattle 5, Wash.
Farrow, Charles Durrett, Jr., KC '54; Hospital of Philadelphia College of Osteopathy, 48th & Spruce Sts., Philadelphia 39, Pa.
Fawcett, Donald L., KC '54; Madison Street Hospital, 1620 18th Ave., Seattle 22, Wash.
Feldman, Raymond K., KCOS '54; Zieger Osteopathic Hospital, 4244 Livernois Ave., Detroit 10, Mich.
Fenwick, Carl H., from 172 Church St., to 58 Alpine St., Oakland, Maine
Fisher, Allen M., from Gilmer, Texas, to Lone Star, Texas
Fisher, Frances Sue King, from Gilmer, Texas, to Lone Star, Texas
Frazier, Robert M., Jr., PCO '54; 158 W. Hortter St., Philadelphia 19, Pa.
Fritch, Edward A., KCOS '54; Carson City Hospital, Carson City, Mich.
Furnish, Richard D., Jr., KC '54; Portland Osteopathic Hospital, 616 N. W. 18th St., Portland 6, Ore.
Ganong, Donald Reade, KCOS '54; Bangor Osteopathic Hospital, 292 State St., Bangor, Maine
Giffen, Larry Arthur, KCOS '54; 416 E. Fillmore, Kirksville, Mo.
Goldenberg, Irving, PCO '54; 1731 N. 57th St., Philadelphia 31, Pa.
Greenspan, Jerome A., PCO '54; 5806 N. 15th St., Philadelphia 41, Pa.
Griffiths, William S., from St. Petersburg, Fla., to 2477 S. Orange Blossom Trail, Orlando, Fla.
Grossman, Mischa F., from 3400 Federal St., to 3084 Federal St., Camden 5, N. J.
Haffenden, James T., from 1331 W. Michigan Ave., to North Avenue Professional Bldg., 170 North Ave., Battle Creek, Mich.
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Karlton, George C., from 1516 E. Los Olas Blvd., to 1221 E. Broward Blvd., Fort Lauderdale, Fla.
Kastning, H. E., from 4167 Lee Ave., to 4903 Delmar Blvd., St. Louis 8, Mo.
Kaye, Stanley B., KC '54; Zieger Osteopathic Hospital, 4244 Livernois Ave., Detroit 10, Mich.
Keefer, Ralph A., from 2207 32nd St., N. W., to 3319 Tuscarawas St., W., Canton 8, Ohio
Kuehlborn, Robert, from Hartland, Wis., to 161 N. 88th St., Wauwatosa 13, Wis.
Kuonen, Martin E., from Bay Village, Ohio, to 4562 W. 130th St., Cleveland 11, Ohio
Lamb, Richard F., from New London, Mo., to 511 N. Florence St., Kirksville, Mo.
Lapham, Hewitt W., from 745 N. Hollywood Way, to 2407 W. Magnolia Blvd., Burbank, Calif.
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Lowe, Amanda L., from Los Angeles, Calif., to Box 94, Sandy, Ore.
Lyon, LeRoy H., Jr., from 2117 Grant Ave., to 4428 Montana St., El Paso, Texas
MacDougall, Howard L., CCO '54; 128 E. 110th St., Seattle 55, Wash.
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Mahan, Glenn W., from Pomona, Calif., to 15600 Pioneer Blvd., Norwalk, Calif.
Marsh, Dorothy J., from Los Angeles, Calif., to 3185 E. Colorado Blvd., Glendale 5, Calif.
Martimick, Andrew, KC '54; 518 Maple Blvd., Kansas City 24, Mo.
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McMains, Harrison, from 1100 W. Yates Ave., to Fort Gatlin Hotel, 545 N. Orange Ave., Orlando, Fla.
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Peterson, C. Gordon, from Clinic Bldg., to 243 W. Third, Albany, Ore.
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Phillips, Ross W., KC '54; 336 Brooklyn Ave., Kansas City 24, Mo.
Pimentel, A. L., from 313 Kauikeolani Bldg., to 1330 S. Beretania St., Honolulu 46, T. H.
Powers, Robert H., from 716 E. Scott St., to 609 E. Jefferson St., Kirksville, Mo.
Pries, Mitchell P., from North Hollywood, Calif., to 13732 Ventura Blvd., Sherman Oaks, Calif.
Randazzo, Michael R., from Northville, Mich., to 3260 Chickering Lane, Bloomfield Hills, Mich.
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Reves, George Oren, from 9971 Garden Grove Blvd., to 12101 Garden Grove Blvd., Garden Grove, Calif.
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Samblanet, H. Louis, CCO '54; 5028 S. Woodlawn Ave., Chicago 15, Ill.
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Scharf, Gerald, PCO '54; 241 S. 47th St., Philadelphia 39, Pa.
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Schwab, J. D. Joseph, KCOS '54; Lakeside Hospital, 2801 Flora Ave., Kansas City 9, Mo.
Schwartz, John P., Jr., from 2822 University Ave., to 603 46th St., Des Moines 12, Iowa
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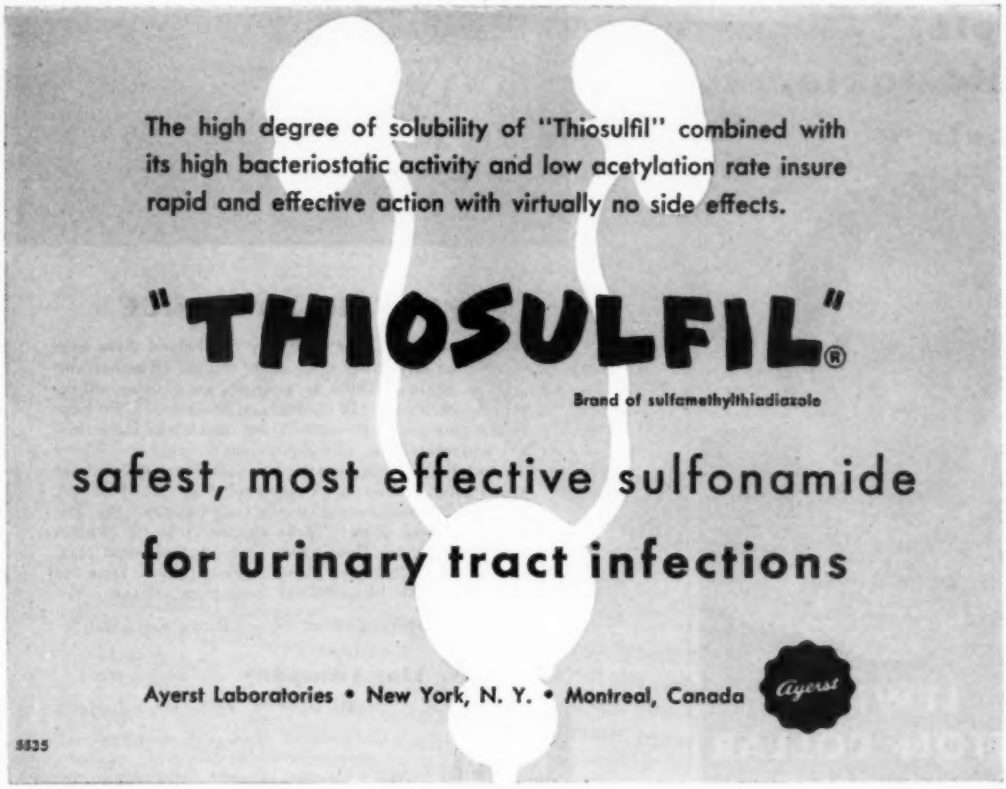
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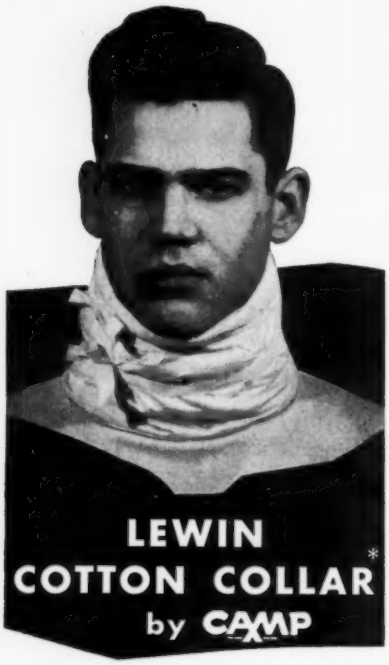

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1. Finnerty, F. A., Jr.: The Value of Rauwolfia Serpentina in the Hypertensive Patient, Am. J. Med. *17*:629 (Nov.) 1954.
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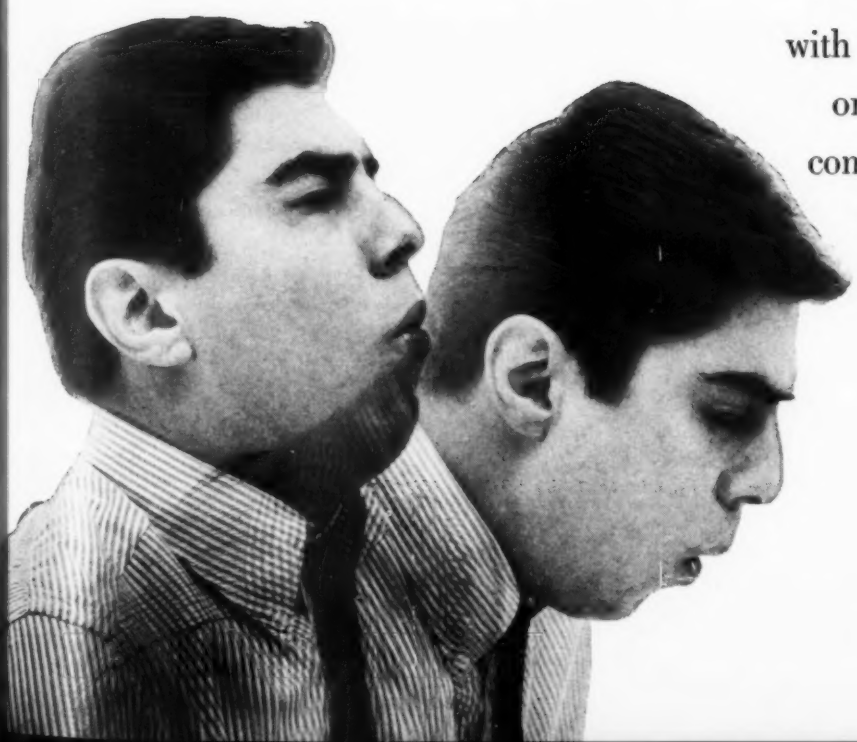

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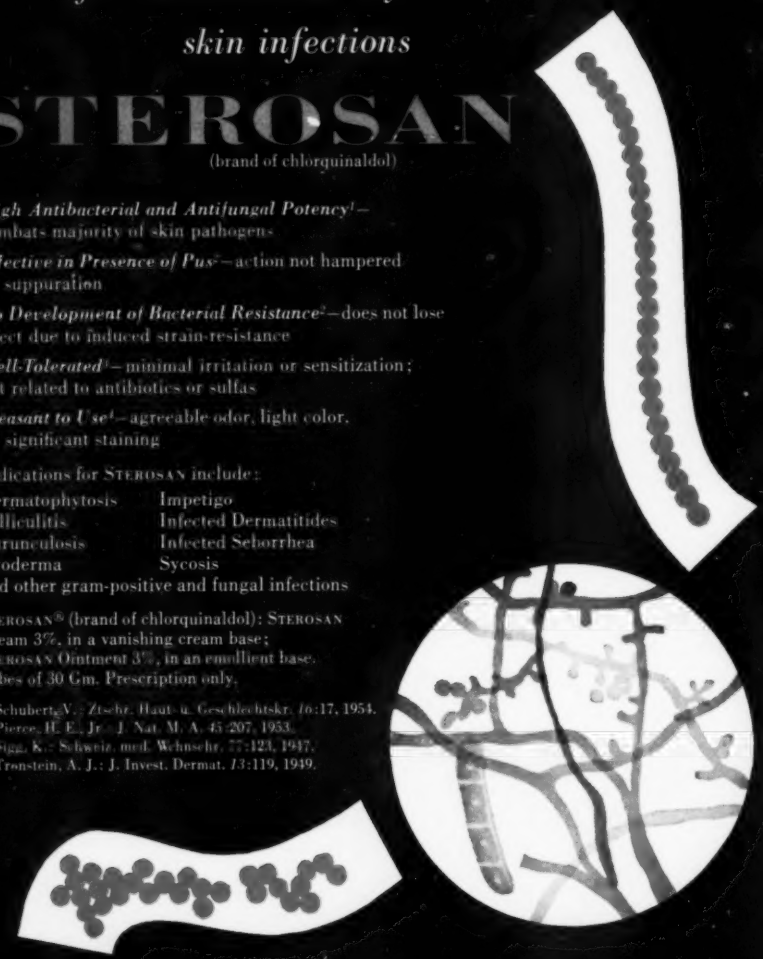

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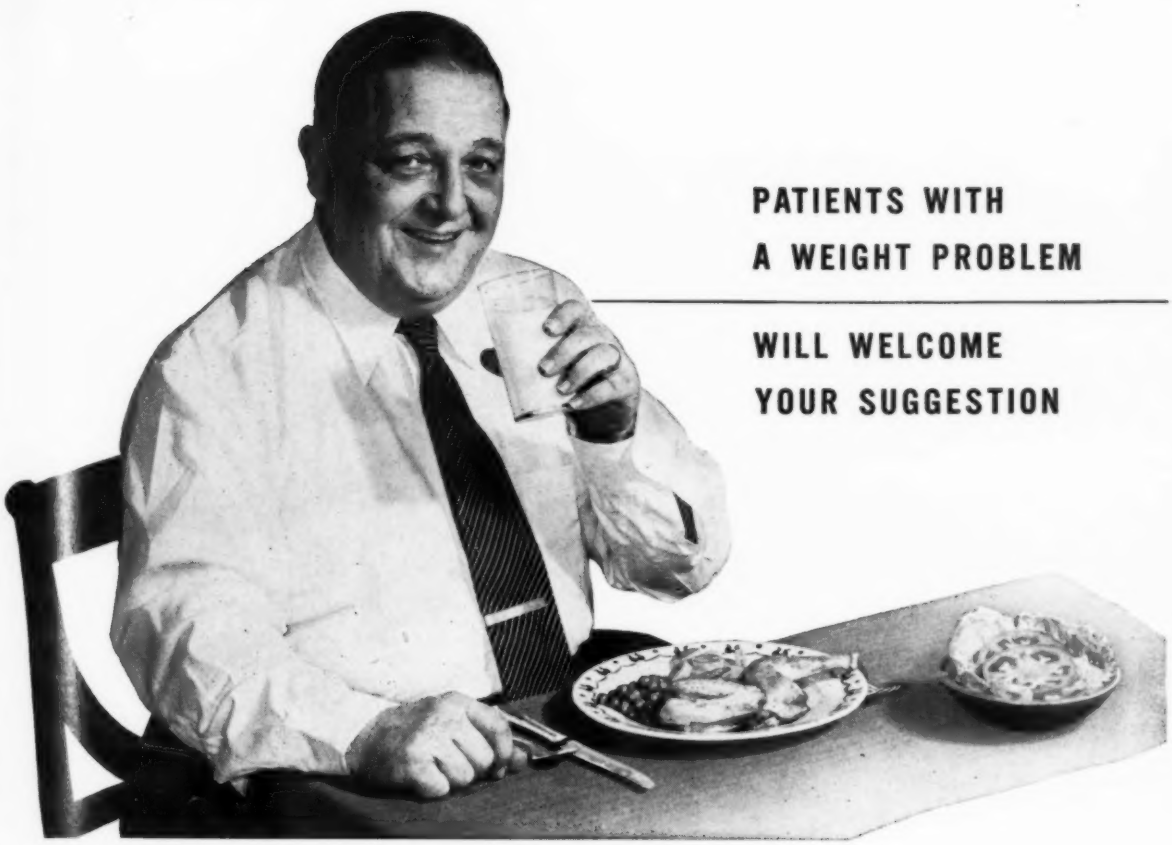

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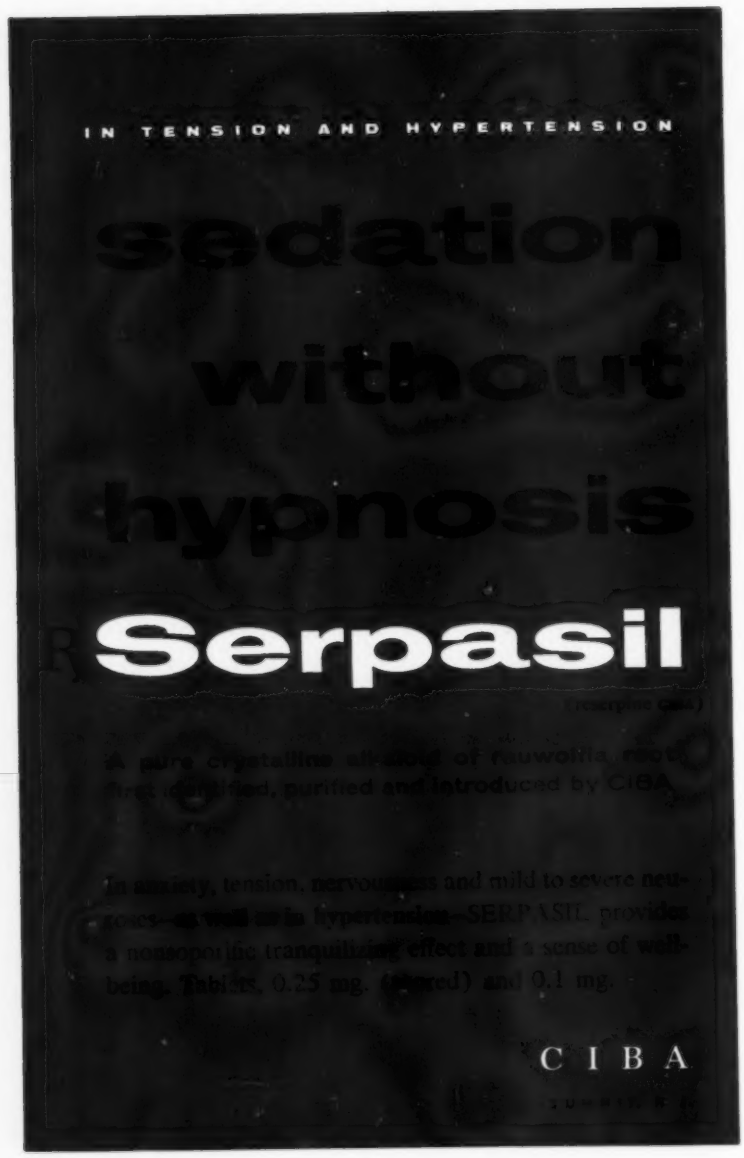
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